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The Journal of Periodontology
is dedicated to
Dr. Gillette Hayden
and
Dr. Grace Rogers Spalding
for their pioneer efforts and
selfless devotion in behalf
of periodontia and the
American Academy
of Periodontology

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Seminar of the American Academy of Periodontology will be held in Ann Arbor, Michigan, April 28, 29, 30, 1952

Thirty-sixth Annual Meeting
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RAYMOND E. JOHNSON, D.D.S.

Treasurer, American Academy of Periodontology

It is not surprising news to announce that Doctor Raymond E. Johnson was elected Treasurer of the American Academy of Periodontology for 1952, since his excellent record has made this selection inevitable. He was first elected Secretary and Treasurer of the Academy in 1936 and served until 1946. He was honored with the presidency of the Academy in 1948 and then the responsibilities of Treasurer were returned to him in 1949.

Dr. Johnson is a respected clinician and teacher of periodontology, serving in the faculty of the School of Dentistry of the University of Minnesota from 1919 to 1948. He has also been active in organized dentistry, serving as Chairman of the Periodontia Section of the A.D.A. (1936), President of the St. Paul District Dental Society (1926), Captain and Unit Commander of Medical Detachment of Minnesota State Guard (1940-45) and is now a member of the St. Paul Area Health Council and Director of the Minnesota Dental Foundation. He is a fellow of the American College of Dentists. His sincere, friendly personality has made Dr. Johnson a most valuable member of the American Academy of Periodontology.

THE JOURNAL OF PERIODONTOLOGY

APRIL, NINETEEN HUNDRED FIFTY-TWO

Observations Concerning the Histogenesis of the Epithelial Attachment*

by LOUIS J. BAUME, D.M.D., M.S., *San Francisco 22, Calif.*

MUCH interest has been directed toward the processes which enact the formation of a shallow sulcus or "normal pocket" between the marginal gingiva and the surface of the tooth. A better understanding of the normal development of the epithelial attachment is a prerequisite for any preventive approach to "pathologic" periodontal pocket formation.

Three concepts have been advanced to explain the development of a normal gingival sulcus: (a) Gottlieb[14] and Orban[20] contend that it results from a separation of the reduced enamel epithelium from the "cuticula dentis," (b) Weski[30] and Euler[12] believe that the sulcus is due to a tear in the epithelial attachment as an "intra-epithelial split," (c) Becks[4] maintains that during tooth eruption the oral epithelium replaces the degenerating enamel epithelium by downward proliferation at its connective tissue side. The bottom of the sulcus is found at the line of fusion of both epithelia. Additional studies with regard to the epithelial changes accompanying tooth eruption have been made by others.[5, 7, 11, 17, 24, 25, 31]

Today it is almost generally understood[15, 21, 29] that the ameloblasts form as their last product the primary cuticle then shorten and degenerate. According to Diamond and Weinmann[8] this involutionary process occurs as soon as the enamel matrix is fully formed even before it is calcified; Wasserman[27] and Marsland,[16] however, describe a prolonged activity of the ameloblasts allegedly in connection with enamel maturation. After the disappearance of the ameloblasts, the remaining epithelial cells of the enamel organ are termed "reduced enamel epithelium" and are ascribed a protective function for the enamel.[21] Proliferation of the reduced enamel epithelium prior to tooth eruption was first mentioned by Meyer[18] in human autopsy material and later confirmed by Nuckolls, Saunders and Frisbie[19] in the rat. While the majority of investigators[14, 15, 20, 31] assumed that the attached reduced enamel epithelium is transformed into an oral stratified squamous epithelium, it was Becks[5] who explained the change as a process of successive replacement.

*From the George Williams Hooper Foundation for Medical Research, the Division of Dental Medicine and the Section of Oral Pathology, College of Dentistry, University of California, San Francisco.

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With the present study it is intended to obtain some additional information on the histogenesis of the epithelial attachment and the formation of the gingival sulcus.

MATERIAL AND METHOD

Since autopsy material of children is rarely available for immediate postmortem fixation necessary for such a study, 20 Rhesus monkeys representing consecutive periods of dental development were used. Their close morphogenetic semblance to the human dental pattern[2] allows for more conclusive evaluation than any other animal material. The lower first permanent molars with their surrounding structures as representatives of the pattern of accessional teeth with no precursors in the deciduous dentition, were selected for vertical and longitudinal sectioning and staining with hematoxylin-eosin as well as according to Masson, Mallory and Koneff. Serial sections provide successive stages of eruption because the mesial cusps always emerge in advance of the distal ones. Permanent central incisors which represent the pattern of successional teeth were sectioned in the vertical or horizontal planes and stained routinely with hematoxylin and eosin. The phase contrast microscope was used for cytological studies since marked contrast can be obtained this way by illuminating the object by a broad light source with a phase retarding plate placed in its geometrical image.[32]

OBSERVATIONS

The histological aspect of the tissues covering an erupting molar is illustrated in Fig. 1. The distal cusps have reached the height of the alveolar crest (B) and the overlying soft tissue is composed of the elements of the enamel organ and the oral mucosa. The oral epithelium exhibits a honeycomb pattern such as described by Emslie and Weinmann[9]. The lamina propria (LP) consists of the papillary zone which is rich in cellular elements and the coarse fiber zone with fewer cells but some larger blood vessels. Below the lamina propria the derivatives of the dental sac (F) have a rather loose appearance, especially in the intercuspal and buccal regions which are not directly opposed to the lingual and vertical eruptive movements of the tooth. The enamel organ still consists of a distinct layer of ameloblasts and a zone of lightly stained prickle cells* with prominent intercellular bridges. At the buccal and intercuspal sites this reduced enamel epithelium extends with slender processes into the connective tissue. Under pressure at the lingual side there are only 2-3 layers of prickle cells and the entire enamel epithelium appears compressed (Fig. 2, EE). Here, the ameloblasts are more cuboidal in contrast to their columnar shape on the buccal side (Fig. 3, Am). At a high magnification of a section stained with iron-hematoxylin-eosin (Fig. 4), a column of dark stained granules is seen to pass through each core of intact slender ameloblasts and determinate with a delta formation in a continuous peripheral membrane (M). The ameloblasts are connected among themselves by delicate intercellular bridges. Their basal ends are in continuity with the prickle cells which contain a dense network of fibrils around an intensively staining nucleus, indicative of high cellular activity. In sections stained with Masson's or Koneff's trichrome and photographed under the phase contrast microscope (Fig. 5) the dark staining intracellular structures are identified as tonofibrils. They enter through the intercellular bridges from the prickle cells and extend aside the nucleus into the dense peripheral membrane which is clearly demarcated from some preparatory residues of the enamel matrix (EM).

Around the tip of the distobuccal cusp (Fig. 6) a change in this arrangement of the enamel epithelium is conspicuous. Between "x-x" the ameloblasts (Am) have

*The term "prickle cell" is used in a mere descriptive way and should not be identified with the prickle cell layer of stratified squamous epithelium (Smith & Copenhager, pg. 379 (26)).

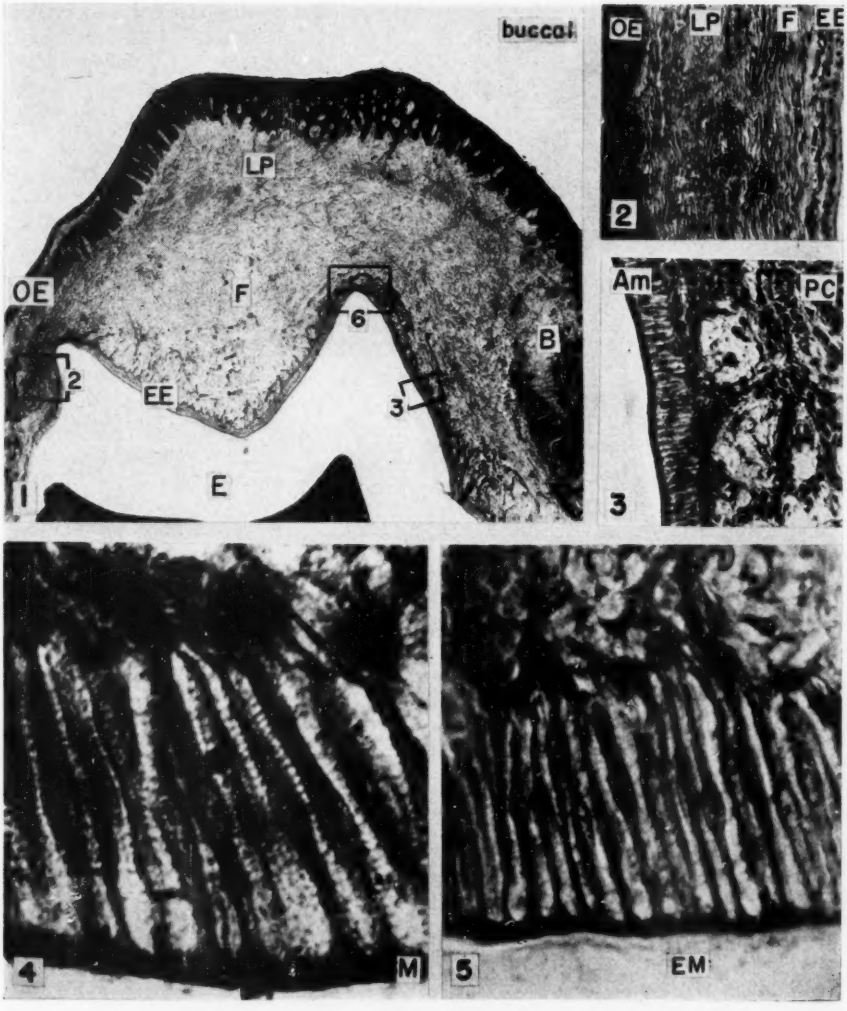

Fig. 1. Buccolingual vertical section through the erupting lower first permanent molar which has approached the lamina propria (LP) of the oral mucosa. OE—Oral epithelium. EE—Enamel epithelium. F—Connective tissue of tooth follicle. B—Alveolar bone. E—Dissolved enamel. Masson, original Mgf. x 25 (Pl. 1525).

Fig. 2. The compressed enamel epithelium (EE) at the lingual side comprises a layer of cuboidal ameloblasts and 2-3 layer of compacted prickle cells. F—Follicular connective tissue. LP—Lamina propria. OE—Oral epithelium. Masson, Mgf. x 130 (Pl. 1578).

Fig. 3. At the buccal side, which is not exposed to horizontal eruptive pressure, the ameloblasts (Am) are slender and protracted and the prickle cells (PC) extend with processes into the loose connective tissue. Masson, Mgf. x 130 (Pl. 1530).

Fig. 4. The formation of a cuticle matrix is revealed by a column of granules passing through the ameloblastic cores continuous with the tonofibrils of the adjacent prickle cells and terminating in an inhomogeneous peripheral membrane (M). Iron H. E., Mgf. x 1200 (Pl. 1572).

Fig. 5. The network of tonofibrils of one prickle cell extends through numerous ameloblasts to the side of the nucleus into the matrix of the enamel cuticle. Note adherent remainders of enamel matrix (EM), Iron H. E., Mgf. x 690 (Pl. 1703). Zeiss phase contrast microscope.

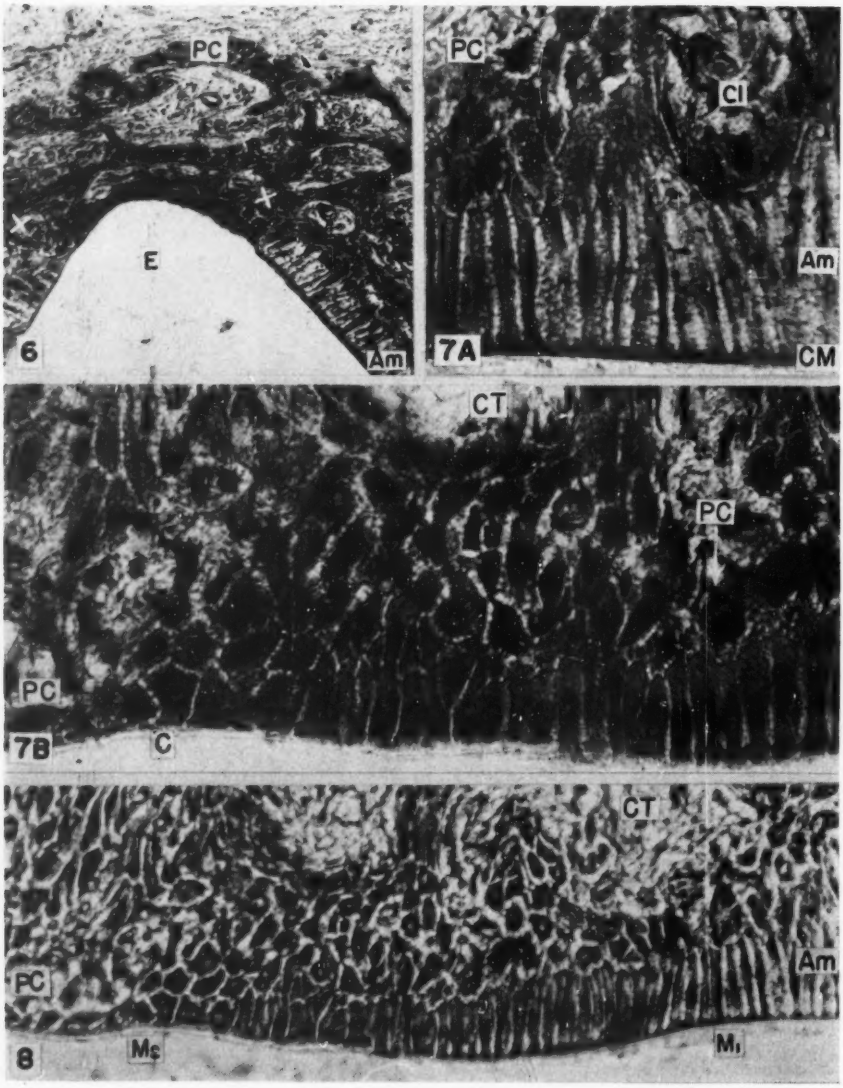

Fig. 6. The enamel epithelium around the distobuccal cusp (E) shows initial stage of involution of the amelobastic layer (Am) concomitant with marked interstitial proliferation (X) of prickle cells (PC). Masson, Mgf. x 130 (Pl. 1530).

Fig. 7A. Intrusion of cluster of prickle cells (Cl) into the amelobiastic layer (Am) signalizing the imminent involution of the latter. CM: Matrix of primary cuticle. Masson, Mgf. x 660 (Pl. 1521).

Fig. 7B. Gradual degeneration and replacement of the amelobiasts by interstitially proliferating prickle cells in the proximal portion of the same section as Fig. 7A. Note decalcified cuticle (C) adjacent to flattened prickle cells. CT—Connective tissue. Masson, Mgf. x 660 (Pl. 1522).

Fig. 8. The calcification of the cuticle matrix (M_1) is revealed by its partial dissolution (M_2) due to laboratory procedure. Koneff, Mgf. x 325 (Pl. 1685A) Phase contrast microscope.

disappeared and are replaced by proliferating prickle cells. Figures 7A and 7B illustrate this successive involution. Clusters of young prickle cells (Fig. 7A, Cl) are seen to penetrate into the ameloblastic layer which displays catabolic changes. The nuclei appear closer to the surface of the enamel, have lost their ovoid shape and are falling into fragments (Fig. 7B). As kariolysis progresses the cytoplasmatic details are lost and the protoplasm exhibits tinctorial changes; concomitantly the cell bodies are seen to become shorter and wider. These changes are always associated with an encroaching proliferation of the adjacent prickle cells. Their nuclei are rich in chromatin and show various phases of mitosis. The arrow in Fig. 7B points to a single prickle cell which penetrates into a group of degenerating ameloblasts connected with them by intercellular bridges. Ultimately the flattened prickle cells completely replace the ameloblasts. A faintly staining membrane, the enamel cuticle (C), is then seen on the enamel surface in continuity with the peripheral membrane of the ameloblast. Figure 8, which was taken under the phase contrast microscope, demonstrates the decreasing density of this cuticle (M_1) at the sites which were more advanced in calcification (M_2).

As the tooth erupts further, marked proliferation of the oral epithelium and a simultaneous lysis of the subepithelial connective tissue takes place, resulting in the ultimate fusion of the oral epithelium and the reduced enamel epithelium (Fig. 9). The basal portion of the epithelium overlying the advancing cusp (10) stains with the same intensity as the entire oral epithelium (OE). Its cytoplasm always shows a strong staining with Masson's fuchsin, while that of the enamel epithelium stains only lightly (EE). The narrow strand of connective tissue between both epithelia shows that the section is taken somewhat to the side of the fusion. Under the phase contrast microscope (Fig. 10) the peripheral layers of the epithelium around the buccal cusp consist of flattened lightly stained prickle cells (PC), many of them displaying kariolysis (K). At "CT" some connective tissue of a high cellular content is encircled. The basal layers (B) of the epithelial complex is occupied by very deeply staining cells. As their nuclei show various mitotic figures they may constitute the germinal layer. In regions of proliferation the more mature cells (OE) show a polygonal form and a round shaped nucleus in an acidophilic cytoplasm. The central cells furthermore contain keratohyaline granules. The staining reaction as well as greater density of their protoplasm identifies these cells as belonging to an oral stratified squamous epithelium. It is noticeable that intercellular bridges are maintained throughout.

Processes of connective tissue lysis are evident in form of granular agglomerations in the coarse fibre zone of the lamina propria (Fig. 9, D). Numerous histocytes (Fig. 11, H) may be seen while the collagenous fibers are losing continuity. In their vicinity ground substance stains basophilic between some degenerating fibrocytes (F) and the remaining fibers. Noteworthy is the hypoemic condition of this tissue. Blood vessels are sparse and small.

As this dissolution of the connective tissue progresses, the oral epithelium grows toward the enamel epithelium. This is demonstrated in a longitudinal section (Fig. 12) through a molar, the mesial cusps of which have pierced the gingiva. The oral epithelium (OE) has proliferated behind the compressed and degenerating enamel epithelium in a honeycomb pattern as far as "A." In the intercuspal area the enamel epithelium (EE) still extends with slender projections into the loose connective tissue of the dental sac. Figure 13 shows a transverse section taken through the erupted mesiobuccal cusp. The buccal portion of the mucosa constitutes the free gingiva while the supracoronal flap progressively is thrust off through exfoliation of the parakeratotic layer of the oral epithelium. The latter is shown in Fig. 14 at a higher

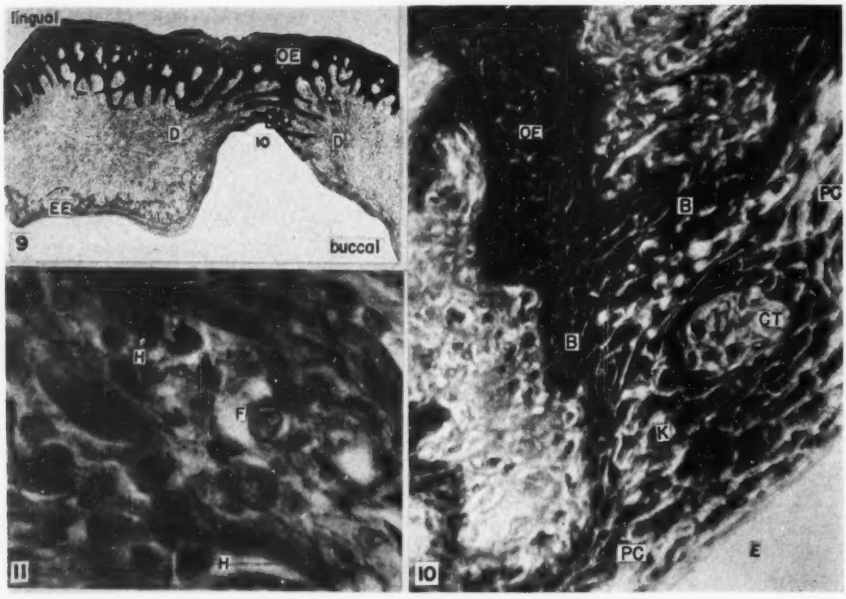

Fig. 9. Buccolingual section through erupting lower first permanent molar shows around the distobuccal cusp (10) deeply staining epithelium which is separated by a small strip of connective tissue from the laterally proliferating oral epithelium (OE). EE—Lightly staining enamel epithelium. D—areas of desmolysis. Masson, Mgf. x 50 (Pl. 1519).

Fig. 10. Epithelial covering of the distobuccal cusp exhibits two types of epithelium elements: The aging prickle cells of the enamel organ (PC) and the proliferating stratified squamous epithelium (OE) with a deeply staining germinal layer (B). Note intensive staining of the cell protoplasm of oral epithelium in contrast to the light staining of enamel epithelium. K—Kariolysis. CT—Connective tissue. Masson, Mgf. x 640 (Pl. 1702) Phase contrast microscope.

Fig. 11. At the sites of desmolysis in the lamina propria (Fig. 9D) agglomerations of histiocytes (H) are recognized. Note lysis of collagen fibre (C), and fibroblasts (F). Iron H. E., Mgf. x 1400 (Pl. 1571).

magnification. The reduction of the enamel organ is proceeding in the same general pattern: replacement of the ameloblasts by prickle cells (A), proliferation of the squamous layers behind the enamel epithelium (B), and disintegration of the prickle cell layers on the enamel surface (C). Here the line of fusion of both epithelia is revealed by the keratinization of the oldest layers of the oral epithelium (K). These processes are also taking place around the lingual cusp and then proceed from both sides toward the center of the gingival covering the occlusal surface. Figure 15 shows this involution at a later phase when the enamel epithelium, including a continuous layer of ameloblasts, is only found in the protected areas of the fissure and the oral epithelium (OE) has advanced toward the center. Due to desmolysis (D) almost the entire lamina propria has disappeared.

At the sites which develop into the free gingiva, the involution of the enamel organ follows a pattern similar to that in the supracoronal portion. Differences are only found in regard to the sequence and timing of events. Figure 16 illustrates the standard pattern. Centers of desmolysis (D) and moderate proliferation of stratified squamous epithelium (OE) at the smooth basal surface of the enamel epithelium (EE) as far as "A" may be seen. The prickle cells beyond this point remain aplastic. At "B" the squamous epithelium is increasing in thickness while the enamel epithelium

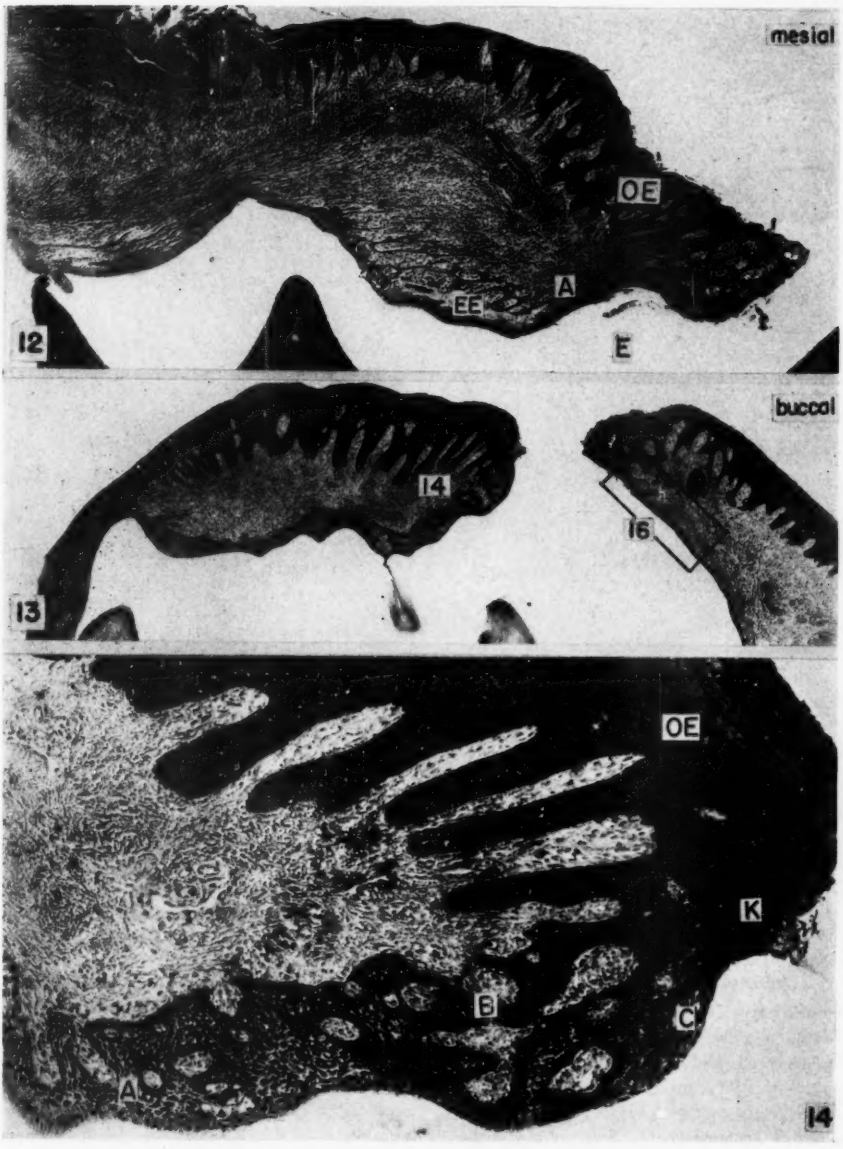

Fig. 12. Sagittal section through lower first permanent molar, the mesial cusp of which has pierced the gingiva demonstrates the honeycomb-like pattern of the fused oral epithelium (OE) with the enamel epithelium (EE) as far as "A." H. E., Mgf. x 30 (Pl. 1554).

Fig. 13. Transverse vertical section through lower first permanent molar showing the mesiobuccal cusp emerged. Masson, Mgf. x 50 (Pl. 1529).

Fig. 14. In the supracoronal flap (Fig. 13) the gradual involution of the enamel organ is seen: At "A" the replacement of the ameloblasts by prickle cells; at "B" proliferation of oral squamous epithelium behind the enamel epithelium; at "C" disintegration of the enamel epithelium; at "K" keratinization of the oldest layers of the oral epithelium (OE). Masson, Mgf. x 130 (Pl. 1536).

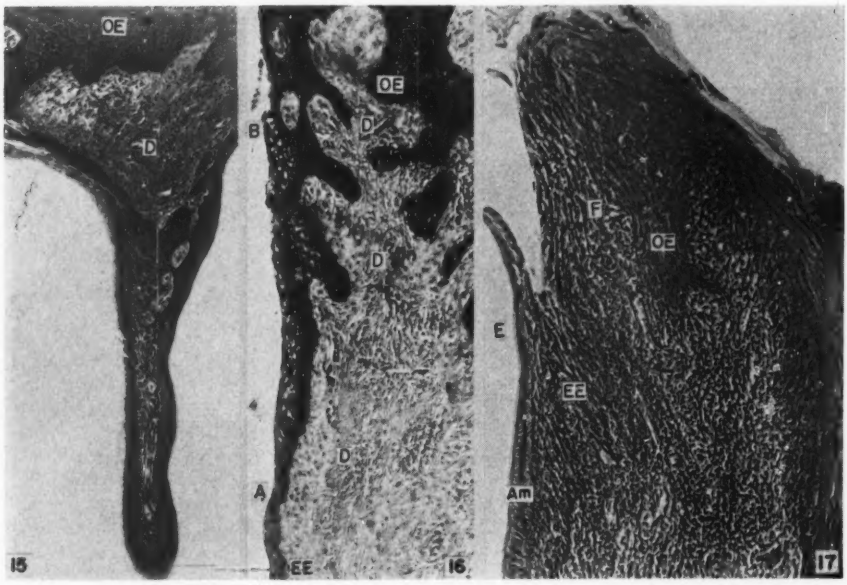

Fig. 15. Remains of enamel epithelium with ameloblasts are found in the occlusal pit when desmolysis (D) has reduced the lamina propria and oral epithelium (OE) has proliferated toward the center. Mallory, Mgf. x 150 (Pl. 1544).

Fig. 16. The standard pattern of sulcus formation is exemplified in the marginal gingiva of Fig. 14. Oral epithelium has proliferated behind the aplastic enamel epithelium (EE) as far as "A." The atrophy of the prickle cells at "B" results in sulcus formation. D—areas of desmolysis. Masson, Mgf. x 290 (Pl. 1542).

Fig. 17. An alternate pattern of attachment is found at the sites where no direct pressure is exerted. Ameloblasts (Am) form the attachment and oral epithelium (OE) proliferates behind the connective tissue of the dental sac (F). Note greater density of the cytoplasm of oral epithelium than enamel epithelium (EE) under phase contrast microscope. H. E., Mgf. x 200 (Pl. 1582).

is seen to undergo progressive aging. A separation and degeneration of these cells along the oral epithelium occurs thereby establishing the gingival sulcus.

Figures 17 and 18 depict a somewhat different normal pattern in which ameloblasts form the attachment to the enamel of an erupted central incisor. The oral epithelium, the cytoplasm of which exhibits greater density under the phase contrast microscope (Fig. 17, OE) than enamel epithelium (EE), grows behind some residues of the dental sac (F). Further apically (Fig. 18) a massive peg is seen to proliferate toward the cervical portion of the reduced enamel epithelium. A tear within the marginal enamel epithelium between the first layers of the prickle cells and shortening ameloblasts is often encountered and may be due to trauma intra vitam or an artifact. This pattern is found at the sites free of pressure such as the distal side of erupting molars and the distolingual of erupting incisors.

Figure 19 illustrates the pattern of attachment in the presence of moderate inflammation (I). Desmolysis is accelerated allowing the oral epithelium to cross the lamina propria with slender projections and to fuse with the cervical portion of the reduced enamel organ. At a higher magnification (Fig. 20) heavily stained oral epithelium (arrow) is seen to proliferate apically along the connective tissue side of the enamel prickle cells. Above the site of fusion the attachment is formed solely by the reduced enamel epithelium (EE).

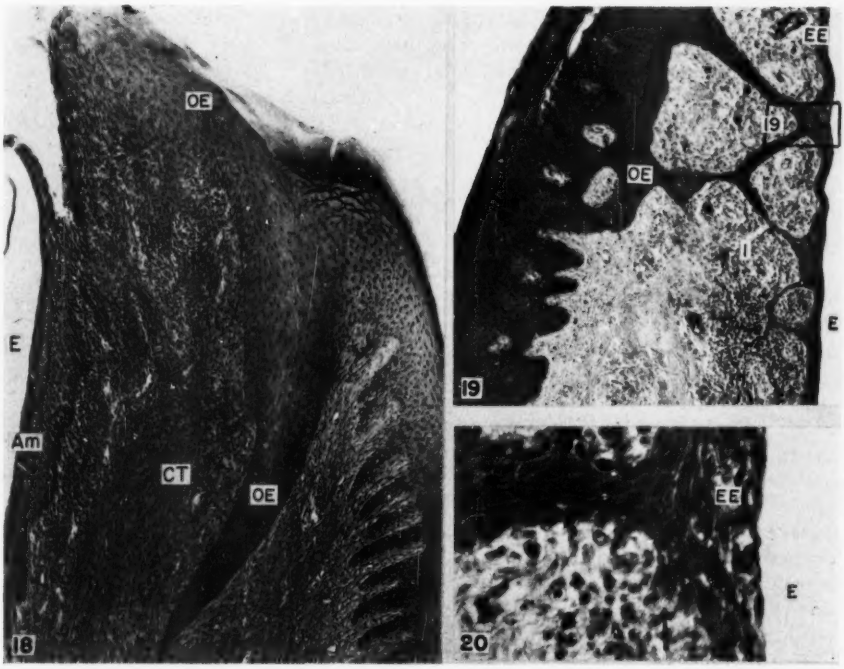

Fig. 18. In the same specimen more apically the oral epithelium (OE) is seen to proliferate in form of a massive peg toward the cervical portion of the enamel organ. H. E., Mgf. x 120 (Pl. 1562).

Fig. 19. In the presence of moderate inflammation (I) the oral epithelium (OE) is seen to extend with transverse projections into the cervical portion of the reduced epithelium (EE). Mallory, Mgf. x 140 (Pl. 1576).

Fig. 20. The connection of the pegs of the oral epithelium with the enamel epithelium is shown at a higher magnification. Note proliferating squamous cells (arrow) behind degenerating enamel epithelium. Mallory, Mgf. x 660 (Pl. 1577).

The sulcus formation in the successional teeth follows the same general pattern as in the accessional teeth. However, the regressive changes in the enamel epithelium can be seen at a much earlier period of tooth development such as demonstrated in an upper central incisor germ at the beginning of root formation. Figure 22 depicts a horizontal section taken about 2 mm. below the alveolar crest. In the area of contact between follicular tissues and the periodontal membrane (A to A) the columnar ameloblastic layer has disappeared. Noteworthy is the marked proliferation of the prickle elements on the opposite lingual side (B). Bone resorption at the vestibular wall of the crypt (O) and opposition at the lingual one are indicative of the forward migration of the germ. In a serial section of a deeper level (Fig. 22) only a few ameloblasts (Am) have been replaced by proliferating prickle cells (PC). Figure 23 represents a vertical section through an erupting upper central incisor. Dark stained squamous epithelium (Fig. 23, A) appears behind the compressed enamel epithelium around the incisal edge in this section which has no connection with the oral epithelium; a large mass of connective tissue intervenes between them. Another serial section of the same tooth (Fig. 24), however, reveals that an actual fusion has taken place between the oral epithelium (OE) and the enamel epithelium further distally. Fol-

lowing fusion, therefore, a rapid lateral spreading of oral epithelium along the connective tissue side of the enamel epithelium already has occurred.

DISCUSSION

The study of the histogenesis of the epithelial attachment to the enamel and the formation of the gingival sulcus revealed various aspects at different sites of the same erupting permanent teeth. The basic processes, however, exhibited a uniform pattern. They consisted of (1) Involution of the enamel organ, (2) Desmolysis of the connective tissue between the epithelia of the enamel organ and the oral mucosa, (3) Fusion of the oral and enamel epithelium. There was variation in the timing and the extent of these processes depending upon the stresses resulting from the preeruptive and eruptive movements. A previous histologic study[3] revealed in addition to vertical migration, medial and forward displacements in the incisors, and lingual and forward thrusts in the molars. Tissues facing these movements are exposed to pressure atrophy and resorption.

1. THE INVOLUTION OF THE ENAMEL ORGAN

After enamel formation a reduction of the outer elements of the enamel organ has occurred. They present themselves as prickle cells and form a syncytium through their intercellular bridges and tonofibrils together with the ameloblasts. Under pressure they become compressed and scarce; this is in contrast to their increased size and number at the opposite sites.

While the tooth starts to erupt and the ameloblasts are still intact, the formation of a cuticle is discernible. In each ameloblast, depending upon the staining method used, there appears a fibril or a column of granules. These intracellular structures are continuous with the network of tonofibrils of the subjacent prickle cells, fuse at the periphery of the ameloblasts and form an inhomogeneous membrane. This matrix is progressively calcified as it was seen to become dissolved by laboratory decalcification at the sites more advanced in development. There, only a structureless cuticle was found. This is evidently identical with the primary cuticle described in the literature.[11, 14, 15, 22]

During or following the formation of the cuticle, the prickle cells begin to proliferate either basally or interstitially apparently related to the eruptive stresses. At the sites free of pressure, it is the basal layer which proliferates markedly and extends with finger-like processes into the follicular tissue while the ameloblasts retain a slender protracted shape.

At the sites under pressure, prickle cells proliferate interstitially, projecting with clusters of new cells toward the ameloblastic column. The basal cell layer grows either moderately or remains aplastic depending upon the environmental conditions. While interstitial proliferation occurs, the initiation of ameloblastic atrophy is recognized. Kariolysis is followed by a shortening and disintegration of the entire cell body. The subjacent prickle cells either indent gradually the basal border line of the degenerating ameloblasts or replace them abruptly. The earliest onset of interstitial cell division is found at the labial side of incisor germs when they begin to form roots. In the molars it is detectable around the mesiobuccal cusp at a much later stage when the tooth penetrates into the lamina propria of the oral mucosa. Stresses of eruption again control this ameloblastic involution since it occurs only in the areas under pressure while on the opposite side the ameloblasts do not yet disappear. There they may form the attachment to the enamel of a tooth which has erupted into occulsion. The end of the life cycle of the ameloblasts thus seems to be more dynamic than previously assumed.

2. DESMOLYTIC PROCESSES IN THE CONNECTIVE TISSUE

In using the conventional histological procedure the study of tissue during tooth

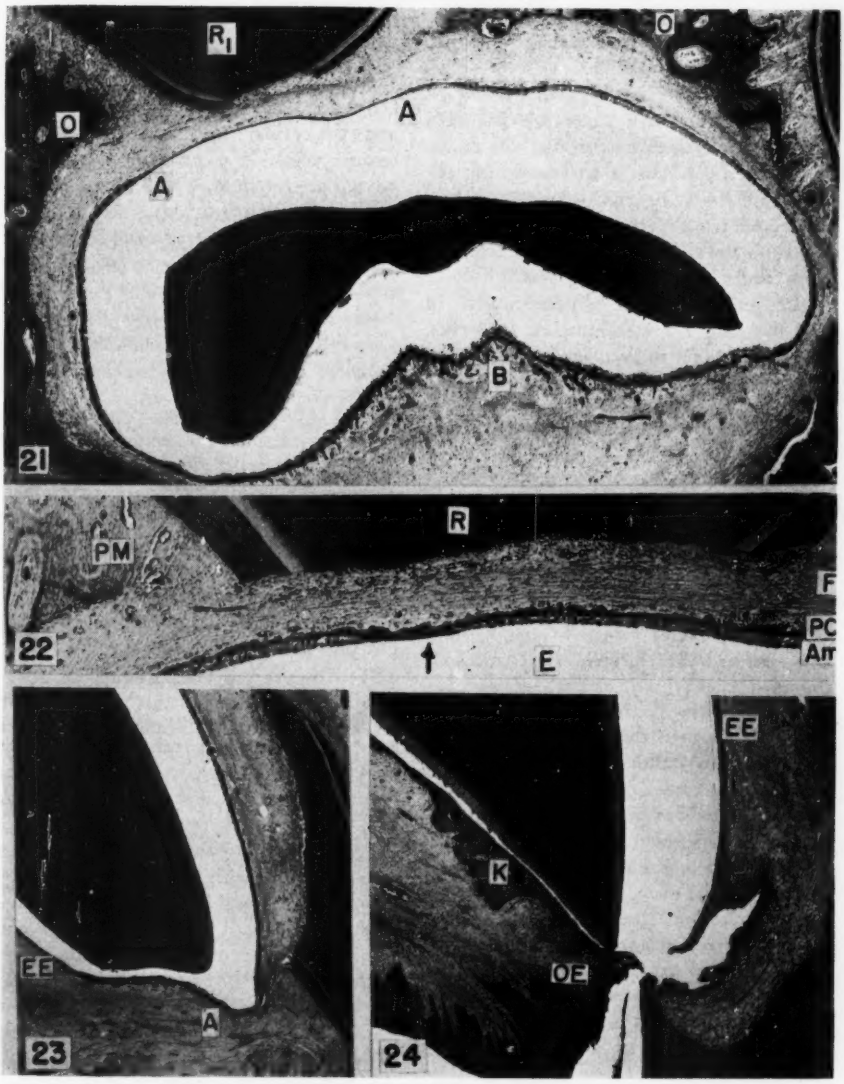

Fig. 21. Horizontal section through the upper central incisor germ at the developmental stage of beginning root formation. Between A and A, which represents the zone of fusion of the dental sac with the periodontal membrane of the predecessor, the ameloblastic layer has disappeared. Note marked proliferation of the prickle cells at the opposite side (B). Osteoclasia in the vestibular wall of the crypt (O) indicates forward movement of the germ. R_1—root of the deciduous central incisor. H. E., Mgf. x 25 (Pl. 1547).

Fig. 22. Horizontal section of the same specimen at a deeper level showing the site of the initial replacement of the ameloblasts (arrow). R—marked resorption at the lingual aspect of the deciduous root. PM—Periodontal membrane. F—Follicular connective tissue. PC—Prickle cells. Am—Ameloblasts. H. E., Mgf. x 60 (Pl. 1549).

Fig. 23. Saggital and vertical section through the erupting upper central incisor behind deciduous homologue showing at "A" deep stained squamous epithelium adjacent to the enamel epithelium. No connection of this squamous epithelium with the oral epithelium is perceptible. H. E., Mgf. x 30 (Pl. 1566).

Fig. 24. Sagittal section taken more distally than the previous in Fig. 22, shows the site of actual fusion of the oral epithelium with the dark stained epithelium to the linguo-occlusal of the tooth germ. Note keratin perls (K). H. E., Mgf. x 30 (Pl. 1568).

eruption must be necessarily restricted to the observations of morphologic changes. Though epithelial changes were predominant, there were distinct indications which suggest a connective tissue role inducing epithelial proliferation. Prior to the fusion of the two epithelia as well as during the epithelial shift, agglomerations of histiocytes and tissue detritus were observed in the lamina propria; simultaneously, collagenous fibres were seen in a state of lysis. These processes occur physiologically. In the presence of inflammation, however, which is known to destroy connective tissue, epithelial proliferation was enhanced considerably.

From observations of tooth retention in amelogenesis imperfecta, Weinmann et al.[28,21] concluded that the reduced enamel epithelium releases an enzyme capable of destroying the connective tissue in order to fuse with the oral epithelium. Ray and Orban[23] in several gingival biopsies, have demonstrated by histochemical methods processes of enzymatic desmolysis some distance from the epithelium. Furthermore, Engle[10] has shown that the connective tissue ground substance overlying the erupting tooth becomes depolymerized before the epithelium has proliferated. Gersh and Catchpole[13] suggest that the action of mucolytic enzymes elaborated by the connective tissue cells may be responsible for this depolymerization. Aisenberg and Aisenberg[1] injected hyaluronidase, an enzyme which liquefied ground substance, into the papillae of monkeys and found that the oral epithelium penetrated with very slender processes deep into the previously injected area.

The observations made in this study also suggest a rather reactive role of the epithelium in desmolysis. Under normal as well as pathologic conditions, an enzymatic destruction of connective tissue elements and the ground substance seems to be the prerequisite for the proliferation of oral epithelium.

3. THE FUSION OF THE ORAL AND ENAMEL EPITHELIUM

A differentiation between the stratified squamous epithelium of the oral mucosa and the prickle cell epithelium of the enamel organ is possible on the basis of tinctorial reactions. The protoplasm of squamous cells has a great affinity to certain acidophilic dyes and therefore appears much darker than that of the enamel prickle cells which behave refractory, especially when stained with Masson's trichrome. This difference is easily recognized under the phase microscope.

Just before a tooth pierces the gingiva, dark stained squamous epithelial cells can be seen around the most advanced portion of the crown. This raises the question as to whether these squamous cells are the direct descendants of the oral epithelium as contended by Becks[4,5] or originate from the enamel epithelium due to metaplastic changes as suggested by Gottlieb,[14] Orban[20] and Westin.[31]

The answer is found in studying serial sections of the early stages of fusion. Whenever heavily stained cells appear to be blended with lighter stained cells of the enamel organ, the oral epithelium always had fused with the enamel epithelium at some place. From there the oral epithelium spreads rapidly with finger-like projections laterally over basal surface of the enamel epithelium. This is well recognized in the supracoronal flap of posterior teeth which is thrust off progressively. At the lateral portions of anterior as well as posterior teeth under horizontal eruptive stresses, the oral epithelium also proliferates along the basal side of the enamel epithelium, even before the crown has erupted. Enamel epithelium remains aplastic and degenerates gradually. As the marginal oral epithelium increases in thickness, the subjacent cells of the enamel epithelium because of insufficient nourishment begin to age and undergo atrophy. A separation and sulcus formation occurs along the oral squamous cells which eventually keratinize. Some

reduced enamel epithelium cells may still adhere to the cuticle at the emerging enamel surface thus constituting a part of the Nasmyth's membrane.[7] This pattern is similar to the description of sulcus formation by Becks and Skillen.

At the sites where no horizontal stresses prevail, an alternate pattern is found. Even when the tooth erupts, the reduced enamel epithelium still consists of typical ameloblasts and several layers of prickle cells. The oral epithelium grows some distance behind the enamel epithelium in a wedge shape apically. It is possible that under functional influences, a tear may occur within the marginal layers of the fragile prickle cells, such as propounded by Euler and Weski. These authors, however, did not describe the alternative and transitory character of this pattern. After further proliferation of the oral epithelium, a fusion with the enamel epithelium occurs later in the deeper cervical gingival portion thereby changing the pattern of sulcus formation.

No evidence is found that the cells of the enamel organ are able to change into a keratinizing stratified squamous epithelium. The appearance of this cell type in the epithelial attachment to the enamel is always a sequence of a proliferation of the oral epithelium from a point of fusion. The change in the histologic composition, therefore, is due to a successive replacement of the aging enamel epithelium by proliferating oral epithelium and not due to metaplasia.

SUMMARY

The histogenesis of the epithelial attachment and the formation of a gingival sulcus was studied in the successional and accessional tooth groups of 20 Rhesus Macaques at consecutive preeruptive and posteruptive stages.

Following the formation of the enamel there occurred a reduction of the outer elements of the enamel organ into a syncytium of uniform prickle cells. The prickle cells were connected with the ameloblasts by intercellular bridges containing prominent tonofibrils. Near the surface of the enamel these tonofibrils were seen to fuse together and to form a continuous membrane, the primary cuticle, which eventually calcified. The epithelial attachment and sulcus formation during tooth eruption were based upon the following histogenic processes: (a) Involution of the enamel organ, (b) Desmolysis of the connective tissue between the oral and enamel epithelium, (c) Fusion of the oral and enamel epithelium. There was a variation in the timing of these events, apparently related to the stresses exerted by the horizontal and vertical movements of the erupting tooth:

(1) At the sites of eruptive pressure the ameloblasts atrophied while prickle cells proliferated interstitially replacing the ameloblasts prior to clinical tooth eruption. As the lamina propria became dissolved, oral epithelium proliferated toward the reduced enamel epithelium, fused with it and grew along the basal layer of the latter. After the tip of the crown had emerged, the reduced enamel epithelium underwent progressive atrophy from the marginal points of increased thickness of oral epithelium thereby establishing the sulcus.

(2) In the absence of pressure prickle cells proliferated with their basal layer in the presence of intact ameloblasts. After eruption of the tooth, the latter formed the primary attachment. Oral epithelium did not fuse continuously with the enamel epithelium, but proliferated first some distance behind it and followed later the pattern described under (1).

Studies with the phase contrast microscope show that even though the two epithelia fuse, the enamel epithelium never changes into stratified squamous epithelium by metaplasia. A process of replacement constitutes the uniform pattern. There was evidence that a normal physiologic desmolysis preceeds proliferation of the oral

epithelium toward the connective tissue side. Cognizance of the enzymatic aspect of this process should offer some new possibilities in the control of excessive downgrowth of the epithelial attachment in pathologic pocket formation and periodontal atrophy.

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(Concluded on page 108)

Mucinase—A Possible Means of Reducing Calculus Formation

by GEORGE G. STEWART, A.B., D.D.S., *Philadelphia, Pa.*

CALCULUS formation is a fairly complex process. There are many opinions about the mechanisms involved. Some authors think primarily in terms of the inorganic precipitants.[1, 9, 19, 30, 33] Others consider the processes of attachment of the inorganic elements of precipitation to the tooth surface as well.[4, 7, 12, 14, 16, 20, 24, 26, 28, 29, 31, 34] However, it is not the purpose of this paper to review that which has been so adequately covered and evaluated by Bibby, Box, Nuckolls, Hodge and Leung, Stones.

It is the opinion of the author that the attachment of the precipitants is of the utmost importance, for without it, calculus, which we recognize clinically, would not exist. The author also feels that mucin plays some role in the attachment mechanism. This view is shared by Prinz, Stones, Hodge and Leung, Savage, Box. However, there is a lack of experimental evidence to substantiate the opinion.

Burnet has described a method of preparing a mucinase or depolimerase which is capable of breaking down mucins of glandular origin. If mucin does play some part in the mechanism of attaching precipitants to the tooth surface, and if this mucin could be altered or destroyed by some medium, such as a mucinase, a reduction in the amount, or change in the composition of the calculus should result.

PROCEDURE

The mucinase was added to the ammoniated dentifrice formula suggested by Grossman.[11] Two batches of this formula were prepared. The first contained 4.65 grams of enzyme to 335 grams of powder; and the second contained twice the amount of the enzyme for the same quantity of powder. Each batch of powder was divided into six equal portions. Compatability studies were made at the laboratories of the Wyeth Institute of Applied Biochemistry.

Six patients, who produced heavy deposits of calculus in relatively short periods of time, were selected from the practice of the author. The original plan of procedure was to thoroughly scale and polish the teeth of these patients and then give them the mucinase containing powder, to use as a dentifrice in the place of any that they had been using previously. They were instructed to brush their teeth in their accustomed manner, without changing either the time or frequency of brushing. Records of the number of days which had elapsed since the last prophylaxis were kept, and an attempt was made to have the patients return for re-examination and prophylaxis at the end of the same period of time.

When the patients returned they again received a thorough prophylaxis, and were given batch number two of the mucinase dentifrice, except when a placebo was given. After the same interval of time the patients were recalled once more and given a thorough prophylaxis. At this time they were told to use a dentifrice of their choice, and were recalled after the given number of days which corresponded to the time of the control period in each case. Both photographic records in color, and written records were kept of each case.

FINDINGS

Only three cases, of the original six, completed the first phase of this study.

Case 1. Mr. W. This patient was observed over a period of 392 days.

Fig. 1-A. Shows the amount of calculus and stain that appeared after 42 days of

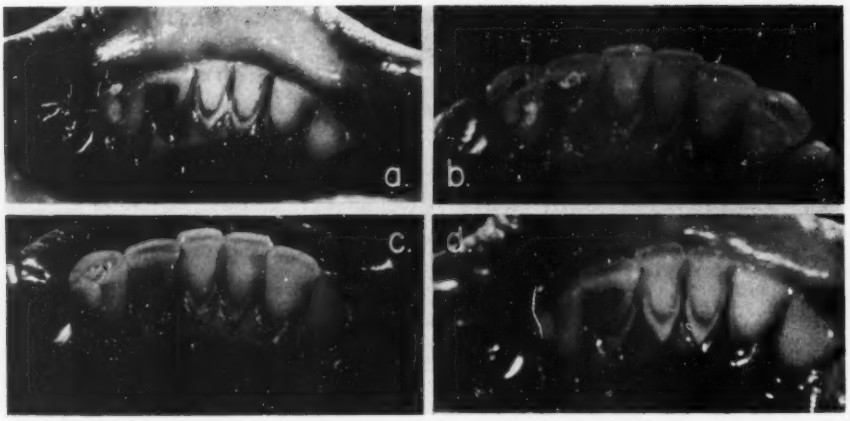

Figure Ia. Mr. W. Preliminary control. 42 days. b. After using mucinase containing dentifrice for 95 days. c. After using Batch II of mucinase containing dentifrice for 135 days. d. "After control"—regular dentifrice used for 120 days.

the control period. He averaged one brushing per day with a regular dentifrice.

Fig. 1-B. After the previous picture was taken and the teeth were cleaned and polished, these are the results after using the dentifrice containing mucinase for 95 days. The teeth were brushed the same number of times a day as above. The calculus that did form during this period was soft.

Fig. 1-C. Prophylaxis was completed after taking picture (Fig. 1-B) and the patient was given a dentifrice containing twice the amount of mucinase that the first batch contained. This was used for 135 days. The calculus which formed was again soft.

Fig. 1-D. This was the after control period, using a regular dentifrice for 120 days. The calculus which formed after this period was hard and glass-like and staining of the teeth was marked.

Case II. Mrs. P. This patient was observed over a period of 227 days.

Fig. 2-A. After a prophylaxis, the patient had used her regular dentifrice for 93 days, averaging two brushings per day. The calculus was hard.

Fig. 2-B. The patient had used the mucinase dentifrice for 43 days, brushing the teeth twice daily. She had returned because the dentifrice had been exhausted. The calculus was almost absent and that which did form was very soft.

Fig. 2-C. A placebo dentifrice had been used for 41 days. The patient had been told that the dentifrice contained twice the amount of the enzyme as that which she had previously used. The number of brushings per day was the same as above. The texture of the calculus was hard.

Fig. 2-D. This picture was taken 50 days after 2-C the patient had been using her regular dentifrice. The calculus was similar to 2-A and 2-C.

Case III. Mrs. C. This patient was observed over a period of 178 days.

Fig. 3-A. The preliminary control period was 40 days after prophylaxis, using a regular dentifrice. The patient had brushed two to three times daily. The calculus was medium-hard.

Fig. 3-B. The patient had used the mucinase dentifrice for 48 days, brushing two to three times daily. The calculus that did

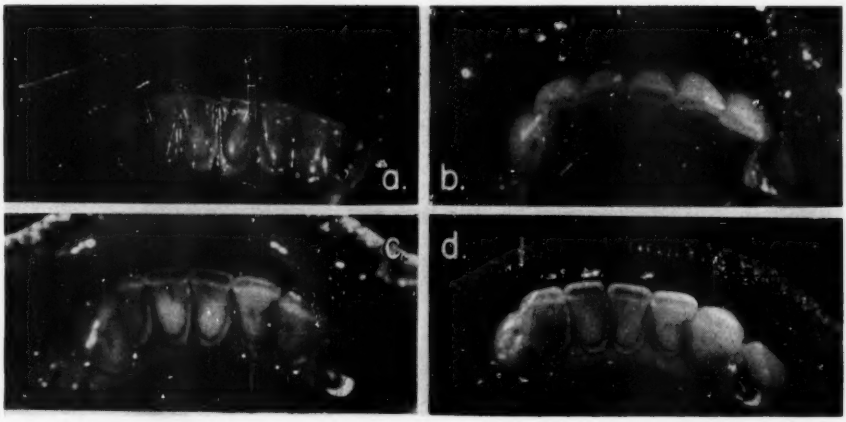

Figure IIa. Mrs. P. Preliminary control. 93 days using regular dentifrice. b. After using mucinase containing dentifrice for 43 days (see text) c. After using Placibo dentifrice for 41 days. d. "After control"—regular dentifrice used for 50 days.

form was soft and crumbly.

Fig. 3-C. After 41 days, with twice the mucinase content, a further reduction in the calculus formation was noted. The calculus was still soft and crumbly.

Fig. 3-D. This was after 49 days using a regular dentifrice two to three times daily. The calculus was quite hard.

The cases studied all showed a definite reduction in the amount of calculus formed when a mucinase dentifrice was used. The calculus that did form, moreover, was much softer than that which appeared when a regular dentifrice had been used, and there was a reduction in the staining of the tooth surface. When the amount of mucinase was increased, there appeared to be a somewhat greater decrease in the calculus formation. There was no reported discomfort or evidence of irritation, and the mucous membranes appeared healthy and pink.

DISCUSSION

The above results seem to indicate that mucin does play some part in calculus formation. This may be attributed to several factors.

Pollard and Smith[23] have shown that algal polysaccharides absorb salts of calcium, magnesium, and iron. Since the physical qualities of these polysaccharides closely resemble those of the gluco polysaccharides of mucins, it would be reasonable to assume that the salivary mucins absorb the precipitated salts of the saliva.

Bacteria present in the saliva could be enmeshed in the mucins from which they could obtain nourishment as has been shown both by Inouye[10] and Dreizen.[13]

The actinomycetes are the predominant organisms found in calculus[9, 27, 28] and Bartels[3] has shown that these organisms can and do produce calcium carbonate crystals in their growth medium. With the initial crystal formation, further crystallization could be expected as was reported by Hodge and Leung[12] and Leung.[18] With the continued flow of saliva, this process could be continued in layers unless some interference by chemical or mechanical means was instituted.

From clinical observation, the author has noted that the heaviest deposits of calculus are found on the lingual surfaces of the lower anterior teeth. These surfaces are closest to the duct openings of the Sub-

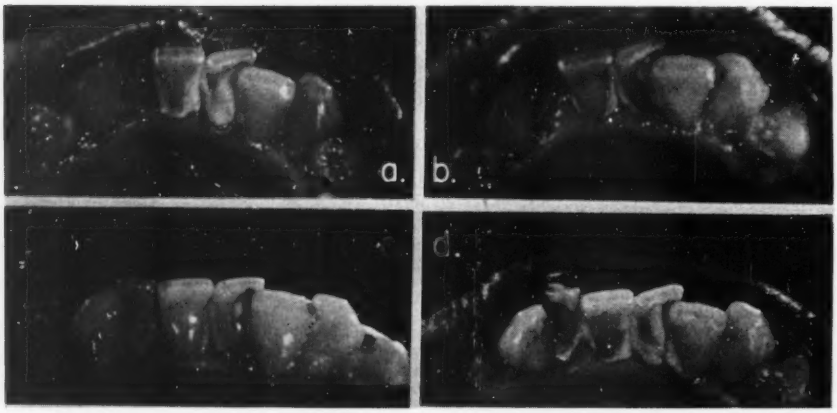

Figure IIIa. Mrs. C. Preliminary control. 40 days using regular dentifrice. b. After using mucinase containing dentifrice for 48 days. c. After using Batch II of mucinase containing dentifrice for 41 days. d. "After control"—regular dentifrice used for 49 days.

maxillary and Sublingual glands which contain the greatest quantities or highest concentrations of mucin.[21] The mucins attached to these tooth surfaces would tend to initiate the above cycle. It would also be closest to the source of the inorganic salts of the saliva as suggested by Leung[13] and so one would expect to find the heaviest calculus deposits in this area.

After a period of time, however, particularly when the oral hygiene is poor, considerable deposits of calculus can accumulate on the buccal surfaces of the maxillary molar teeth as well. This is felt to be the next greatest area of mucin concentration since the parotid gland, although primarily a serous secreting gland, does produce albumins which are adhesive.[21] They also secrete some mucins according to Bremer,[5] who states that goblet cells are present in the secretory portion of the Parotid Duct. The mucins thus secreted could, by the previously described mechanism, aid in the formation of calculus in this area.

Since oral fluids are constantly moving about the mouth during speech, swallowing, etc., as was shown by Appleton and Williams,[2] the oral mucins along with the bacteria and inorganic salts, are probably carried to all corners of the mouth. These substances become adherent to the tooth surfaces and initiate the cycles described. In those areas where the routine methods of oral hygiene or the regular friction of chewing, fail to disturb them, we find heavy deposits.

We may then reason that the glandular mucins in the mouth, which have been depolymerized by the mucinase can probably no longer hold the bacteria as they normally would. Also the mucins which would ordinarily be a source of nourishment to the actinomycetes and a medium in which the deposition of crystals by these organisms could take place, would be substantially reduced, and so the matrix for the precipitated inorganic salivary salts would be less efficient, and the calculus that would form would be scanty and not as hard. This was shown to be true in the clinical findings.

Other methods for reduction of calculus formation have been proposed. These methods were designed to dissolve the inorganic elements in calculus. Kerr proposed Sodiumhexamasphosphate, which was effective and did not harm the tooth surface. When silicate restoratives were pres-

ent, this compound was contraindicated, because it would dissolve them. Stones has reviewed many acid preparations for dissolution of the inorganic salts of calculus but concludes that they may be harmful to the tooth structure.

Further studies are now in progress both clinical and microscopic to study the effect of mucinase on calculus formation and the mucins and bacteria therein. Knowledge thus gained may be useful in reducing calculus formation and in the prevention and treatment of periodontal disease.

The author wishes to thank Dr. Joseph Seifter and Dr. George H. Warren of the Wyeth Institute of Applied Biochemistry for the mucinase powder and the compatability studies.

SUMMARY

1. Mucinase, when added to a compatible dentifrice reduced the amount of calculus formed in the cases reported.

2. The calculus that did form was much softer than that found when a regular dentifrice was used.

3. The patients experimented no discomfort when using the mucinase containing dentifrice and the gingival tissues were firm and pink.

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Study of Cervical Radiolucent Areas in Human Teeth

by BALBIR S. MADAN, D.D.S. AND FINN BRUDEVOLD, D.D.S.,* *Rochester, N. Y.*

FREQUENTLY, radiolucent areas, which cannot be associated with any known pathological condition, are encountered in the cervical region of teeth. These areas are usually found extending apically from the cemento enamel junction, but they may also, in some cases, extend a short distance into the coronal part of the tooth. The areas have not been seen to extend apically beyond the crest of the alveolar bone. In most instances they appear as curved elongated shadows with the convexity toward the pulp. They vary in depth and may occasionally extend as far as the pulp. These areas may or may not have a sharp outline. One of the characteristics is that the profile of the tooth usually appears undisturbed because there is no loss of tooth substance. From a practical point of view, these radiolucent areas often represent a diagnostic problem because they may be difficult to distinguish from radiolucent areas associated with dental caries.

Little attention has been given to these radiolucent areas in the literature. Raper[1] and Durbech[2] considered them to be the result of concavities and convexities present in the teeth; the X-ray beam having to penetrate corresponding differences in bulk of tooth structure. Riffle[3] made the interesting observation that these radiolucent areas occurred frequently in persons with periodontal disease, and he believes they may be related to periodontal pathology. He found that the areas on the root corresponding to the radiolucencies could be curetted more easily with sharp instruments than the rest of the root and, therefore, believed that the radiolucent areas demonstrated a pathological softening of tooth structure.

In reviewing a series of X-rays, we found that the cervical radiolucent areas appeared to be more prevalent in older than in younger persons. This observation seemed sufficiently interesting to undertake the present study, the purpose of which is to obtain information about the occurrence and extent of these areas in different teeth of different age groups. Also, an attempt is made to establish the nature of the radiolucent areas by X-ray examination of skulls and extracted teeth.

OCCURRENCE OF CERVICAL RADIOLUCENCIES

Dental roentgenograms which had been taken of a group of industrial workers in

*Eastman Dental Dispensary.

TABLE I

UPPER TEETH

Age Group	*Right*							*Left*						
	7	*6*	*5*	*4*	*3*	*2*	*1*	*1*	*2*	*3*	*4*	*5*	*6*	*7*
20 - 29	3.3	6.4	1.0	2.8	3.8	4.3	4.2	5.3	4.5	4.2	3.1	1.4	6.0	4.1
30 - 39	4.2	7.8	2.7	3.8	4.9	5.2	7.7	5.1	5.5	5.8	3.9	2.8	8.1	3.8
40 - 50	9.0	8.9	5.6	6.6	6.8	6.3	11.6	13.3	10.5	4.6	7.2	6.1	9.4	8.5

LOWER TEETH

20 - 29	1.8	3.8	2.1	4.2	4.7	4.3	3.8	3.4	4.7	4.8	4.9	4.3	5.9	4.5
30 - 39	3.7	7.2	5.7	6.1	5.7	7.2	7.0	6.6	6.5	7.3	6.1	4.7	5.6	4.8
40 - 50	5.5	8.6	8.1	8.1	8.9	9.2	6.8	8.5	10.3	9.5	9.4	7.1	8.9	9.8

MEAN AREAS OF CERVICAL RADIOLUCENCIES IN INDIVIDUAL UPPER AND LOWER TEETH

The Figures Indicate Number of Squares Covered by the Radiolucent Areas

connection with routine dental examinations were examined for cervical radiolucencies. Only cases with a full set of clear roentgenograms* were included. An enlarged image of the roentgenogram was obtained by projecting it under standard conditions on square graph paper (each square 90 mm.^{2}). The area of the radiolucencies was measured by counting the number of squares they covered. All teeth were examined for radiolucencies except the third molars, which were excluded, because they were not clearly reproduced in most of the roentgenograms. A total of 107 full mouth roentgenograms were studied. These were divided in the following age group: 20 to 29 years, 31 cases; 30 to 39 years, 38 cases, and 40 to 50 years, 38 cases.

RESULTS

Table I shows the mean area of radiolucency found on individual teeth in each age group. This was calculated by adding the areas found on the different types of teeth and dividing by the number of cases examined. There is a definite increase with age in the extent of cervical radiolucencies in both maxillary and mandibular teeth. For most teeth the increase was considerably greater in the 40 to 50 years age group than in the younger groups.

The distribution of radiolucent areas tended to be symmetrical in the upper and lower teeth, but different teeth were involved to different extents. This was particularly true for the upper teeth. In the two younger groups the upper first molars showed the largest cervical radiolucencies and the bicuspids the smallest. In the oldest age group the cuspids and the bicuspids showed the smallest cervical radiolucencies. No pronounced difference was noticed in the extent of the radiolucent areas of the mandibular teeth.

In Graph I the sums of the mean radiolucent areas of the maxillary and mandibular teeth for each age group are compared. There appears to be no difference

*The roentgenograms were provided through the courtesy of Dr. A. Porter S. Sweet of the Eastman Kodak Company.

TABLE II

PERCENTAGE OF TEETH SHOWING CERVICAL RADIOLUCENT AREAS

UPPER TEETH

Age Group	Right							Left						
	7	6	5	4	3	2	1	1	2	3	4	5	6	7
20 - 29	63.4	70.3	35.5	57.8	59.9	64.3	76.1	78.0	73.0	65.0	44.6	51.5	77.9	60.7
30 - 39	74.9	79.7	60.6	58.7	67.6	78.3	86.0	84.0	88.5	77.1	74.4	61.1	90.0	78.2
40 - 50	85.4	87.2	51.2	80.3	62.3	77.2	92.3	92.8	96.0	88.8	77.3	65.3	95.6	91.2

LOWER TEETH

20 - 29	63.4	70.3	35.6	58.0	55.0	64.3	75.0	78.0	73.0	65.0	44.0	53.5	77.7	61.7
30 - 39	65.6	86.1	70.3	80.0	67.1	82.3	81.3	82.8	83.7	91.8	73.3	60.5	81.0	82.0
40 - 50	68.1	82.5	82.1	81.6	74.2	82.3	65.6	72.2	86.2	83.8	86.3	76.7	85.0	83.2

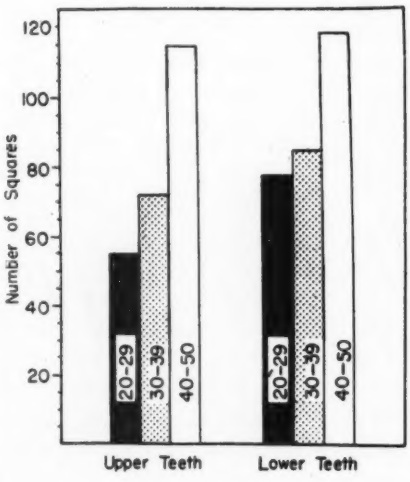
except for the group 20 to 29 years which shows the mandibular teeth to be more involved than the maxillary teeth.

The percentage of teeth in each age group which was found to have cervical radiolucencies is shown in Table II. Again there is a definite increase with age. The bicuspids are, generally, least involved in the youngest age group. In the older age groups the teeth are more evenly affected.

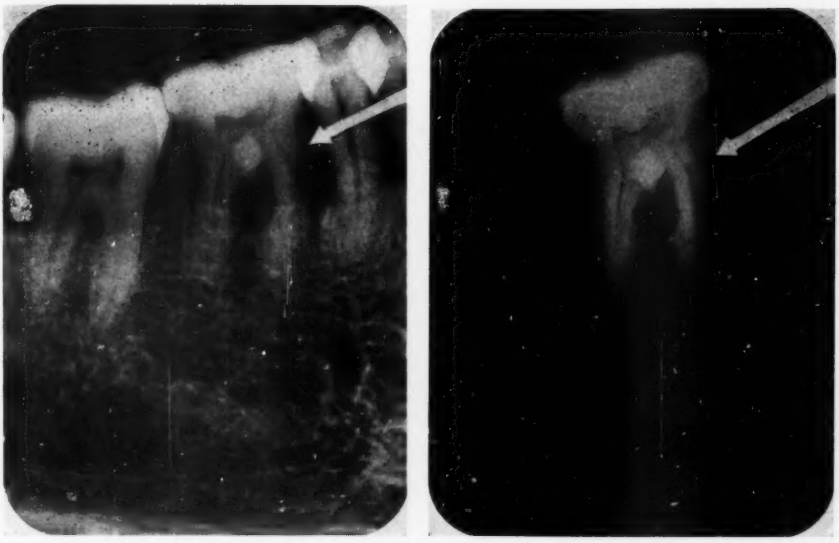
EXAMINATION OF RADIOLUCENCIES IN EXTRACTED TEETH AND TEETH IN SKULLS

Roentgenograms were taken of extracted teeth using different angulations. The X-ray tube and the film were kept in a fixed position, but the teeth were rotated a few degrees between each exposure. Using this technique it was found that cervical radiolucencies could be produced at will and that they were the result of convexities and concavities in the teeth as suggested by Raper. In a few instances it was possible to examine extracted teeth which showed cervical radiolucency in roentgenograms taken before the extraction. The radiolucent areas could be approximately reproduced when the teeth were exposed employing the same angulation as used in the pre-extraction roentgenograms. Again, the areas could be explained by curvatures in the anatomy of the root.

Although these findings demonstrated that the cervical radiolucencies were caused by convexities and concavities in tooth anatomy, they did not explain the increase



Graph I
Sum of mean radiolucent areas of upper and lower teeth in different age groups.



Roentgenogram of a dried mandible at the left shows a radiolucent area terminating at the alveolar margin. Roentgenogram at the right shows the same tooth removed from the alveolus and exposed in the same position. The radiolucent area is seen extending to the apex. (The area at the bifurcation is an artifact produced because the root had been chipped.)

in radiolucent areas with age. It was suspected that the presence of alveolar bone around the root might obscure or obliterate the presence of radiolucent areas. This possibility was investigated by taking roentgenograms of teeth in skulls which had various degrees of loss of alveolar bone. The position of the X-ray tube and the film was stationary and the skull was placed in such a position that cervical radiolucent areas were produced in the X-ray image. The tooth which showed cervical radiolucency was then removed from the skull and arrangements were made to take a radiograph of this tooth in the same position with respect to the film and the X-ray tube as it had in the jaw. This was achieved by taking a compound impression of the teeth. After the position of this impression had been fixed, the skull was removed and the tooth to be studied was replaced in the impression and X-rayed.

From these experiments it was found that the shadow terminated at the alveolar ridge when the tooth was in the alveolar socket, whereas, the radiolucency could be seen extending further apically when the tooth was X-rayed out of the mandible.

It was concluded that the increase of cervical radiolucency with age resulted from the increasing loss of alveolar bone in the older age groups.

DISCUSSION

It was found that most of the teeth examined in the present study had cervical radiolucent areas. This may be logically explained by the observation that these areas are associated with curvatures in the anatomy of the root. With the irregular shape commonly found in teeth, there will be, in most instances, appreciable difference in the amount of tooth structure which the X-ray beam penetrates when roentgenograms are taken.

Perhaps the most interesting observation in this study is the finding that these areas

increase and occur more frequently with age and that they are related to the level of alveolar bone. The fact that the alveolar bone obliterates the radiolucent areas on the root so that only the radiolucent area above the alveolar crest appears on the roentgenograms gives our data the added significance of revealing in a quantitative way, the loss of alveolar bone which tends to occur with age.

Of course, the presence of a cervical radiolucency is not always associated with bone loss. This is demonstrated by the fact that these areas were found in more than half of the cases examined in the 20 to 29 years group and the not uncommon presence of these areas in roentgenograms of children. Nor does the absence of cervical radiolucencies exclude the possibility of alveolar bone loss. What has been demonstrated is that radiolucent areas which extend apically along the root are made visible only when there is alveolar bone loss. Therefore, it is believed that the increase in the extent of the cervical radiolucencies which was found with age, gives a fairly accurate picture of the decline of the supporting structure of the teeth. According to this concept, our findings show that there is continuous and progressive increase in loss of alveolar bone with age, particularly noticeable in the 40 to 49 years group.

Marshall-Day[4] seems to be the only investigator who has attempted to make a quantitative evaluation of bone loss in a population group. He also found increased bone loss with age, but his data, obtained in India, are not comparable to ours because of the much higher incidence of periodontal disease in that country. Black[5] studied the incidence of alveolar bone resorption. He found that alveolar bone loss occurred in about twenty percent of persons 20 to 29 years of age and that the incidence increased rapidly in older groups. This is in general agreement with the present observations.

It is of interest that many clinicians believe that a pathological softening may be detectable in the cementum and even in the dentin of teeth affected by periodontal disease. This possibility is not ruled out by the present work. An answer to this problem will require examination of more teeth and the use of other experimental procedures than those we used. No evidence has been found in the literature showing that the radiolucent areas actually may represent softened tooth structure. Simpson[6] seems to be the only worker who has attempted to determine the changes which may occur in cementum of teeth with periodontal disease. He found that cementum from teeth with periodontal involvement had smaller phosphorus and higher calcium content than cementum of teeth without periodontal involvement. This change, if at all radiologically detectable, would be expected to produce a radio-opaque and not a radiolucent area.

More information is needed about the changes of cementum in periodontal disease. Chemical and histochemical studies in this area might contribute to our knowledge of the nature of periodontal disease.

It is concluded from this study that tooth curvatures are associated with radiolucent areas in the cervical and root portion of teeth and that the extent of the radiolucent area may depend on the alveolar bone level. The problem of distinguishing these areas from radiolucent areas caused by dental caries remains to be a challenge to practicing dentists.

SUMMARY

The extent of radiolucent areas in the cervical and root portion of teeth was studied in 107 sets of full mouth roentgenograms. The areas occurred more frequently in older than in younger persons and the size of the areas increased with age. The areas were found to be produced by differences in bulk of tooth structure with associated difference in X-ray penetration.

It was demonstrated that alveolar bone obliterated the appearance of the radio-

lucent areas. Dental roentgenograms taken of the jaws of skulls showed radiolucent areas extending to the margin of the alveolar bone. When the teeth with radiolucent areas were removed from the socket and X-rayed, the areas could often be found to extend further apically. For this reason the extent of radiolucent areas in different age groups was considered related to loss of alveolar bone.

Grateful acknowledgement is made to Dr. L. V. Lopez, Manager and Dr. David Lempert, Chief of the Dental Division of the Veterans' Hospital in Canandaigua, New York for providing facilities for making preliminary observations and to Dr. A. Porter S. Sweet of the Eastman Kodak Company, Rochester, New York for providing roentgenograms used in this study.

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The Permeability of Human Dental Root Structures to Radioactive Iodine*†

by WILLIAM WARD WAINWRIGHT, D.D.S., M.S., *Chicago, Illinois*

MANY questions confront the periodontist regarding the possibility of cementum protecting itself against disease. Among the many factors known to be involved, a prominent one is the permeability of the cementum. Can physiologic processes within the cementum aid in healing damage to the tooth supporting structures either before or after its exposure by pocket formation? Can cementum pass nutrient material, toxins, fluids, salts, etc.? Information on these questions has thus far largely been dependent upon visual observation of: the living tooth and the attached soft tissues, the exposed cementum after recession of the gingival tissues, and, after extraction, dialysis experiments and the microscopic appearance of ground and decalcified sections. The only means with which to check such visual observations has been the response of exposed cementum and dentin to irritation. One is often so impressed with the complaint of the patient, that the exposed neck of the tooth is sensitive, that one applies caustics and similar materials to relieve the symptom. Although this is a necessary

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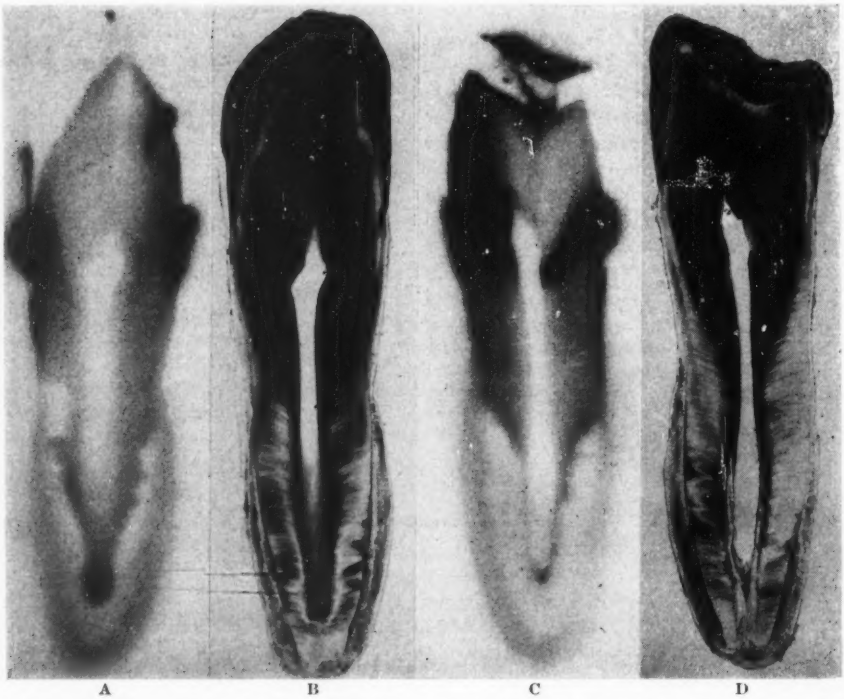


A B C D

Fig. 1. Radioautographs (A & C) of ground sections (B & D) from upper left second bicuspid extracted five hours after administration of 25 mc. I^{131} to a 33 year old male.

clinical procedure it is readily appreciated that the deadening effect can only result in the loss of vital functions of the structures treated. In a sense this seems a contradiction of our effort to preserve the teeth.

Hints of more far reaching factors have appeared from time to time in the literature. For example, the findings of Cahn (1926), Bass (1951) and Riffle (1951) present evidence of changes in dentin and cementum which has become exposed to the mouth.

Stones (1948) has summarized information on the permeability of cementum. He notes that Sharpey's fibers when embedded in the cementum are fully calcified and the resulting acellular cementum, except in the very young, becomes completely impermeable to dyes. He concludes further that this cementum is also impermeable to lymph. He concludes also that there are no channels along which micro organisms may penetrate, contrary to the situation in the tubules of the dentin or in the cellular cementum of the apical portion of the root.

A distinct impetus to investigations of the permeability of dental structures has been provided by the increasing number of dental investigations with radioactive materials. Bartelstone (1950) reviewed the history of work in this field and in summarizing his own investigations emphasized the importance of examining human material, which is readily available where the teeth are to be extracted from patients treated

Fig. 2. Radioautographs (A & C) of ground sections (B & D) from upper left lateral incisor extracted 17 hours after administration of 25 mc. I^{131} to a 43 year old male.

with radioactive iodine for thyroid disease. He found many new observations in conjunction with diseases of the periodontium. Repeatedly he found that exposed cervical dentin whether metamorphosed or not was readily permeated by this tracer. Even the secondary dentin at the end of the metamorphosed tracks was penetrated, contrary to the earlier findings of Stones and others who used dyes as tracers. Bartelstone also found the cementum penetrated not only from exposed surfaces but also from the normal periodontal structures beneath the gingival crevice and from the dentinal side via the pulpal circulation.

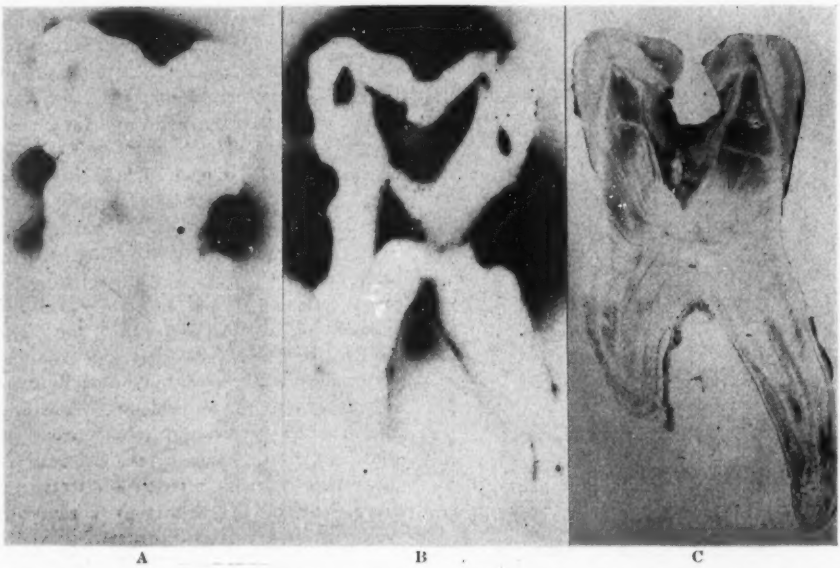
The effectiveness of the radioactive tracers in periodontal investigation is demonstrated well by the current work of Götte, Hattemer, and Frimmer of the University of Mainz, Germany. In an investigation of the possible penetration of arsenic beyond the pulp tissue during devitalization, radioactive arsenic (As^{76}) was used in the form of the trioxide. Radioautographic evidence was found of a toxic concentration of arsenic in the cementum in as short a time as two to four days. In fact such large quantities of arsenic penetrated the tooth that of the arsenic remaining after removal of the original arsenic tablet only 1% was found in the pulp proper and 99% had migrated into the surrounding dentin and cementum. The investigators conclude that this amount of arsenic in the cementum must be considered as a source of prolonged damaging effects upon periodontal structures. The illustrations accompanying the article make quite clear the wide distribution of arsenic from the point of application to the pulp on to the dentin, cementum, and enamel.

Experiments with radioactive plutonium citrate, radioactive thiourea, and stable silver nitrate indicate that there are at least three levels of permeability of enamel (Wainwright 1951 A & B). Further investigation of the permeability of cementum by the methods of Bartelstone were made possible through the cooperation of the Staff of the Veterans' Administration Hospital, Hines, Illinois.

METHOD

When teeth were to be extracted from patients receiving radioactive iodine (I^{131}) the extractions were scheduled at the indicated periods after the administration of I^{131}. The teeth were immediately sectioned under oil. Radioautographs were made by pressing the ground sections against fast dental x-ray film. The film is exposed by the radioactivity of the iodine thus disclosing the location and relative amounts of iodine present.

Since I^{131} loses its radioactivity rapidly ($t\,½ = 8$ days) radioautographs of equal exposure, however made at different times, cannot always be compared as to density. Comparisons can be made if the exposure time is corrected for radioactive decay.



A B C

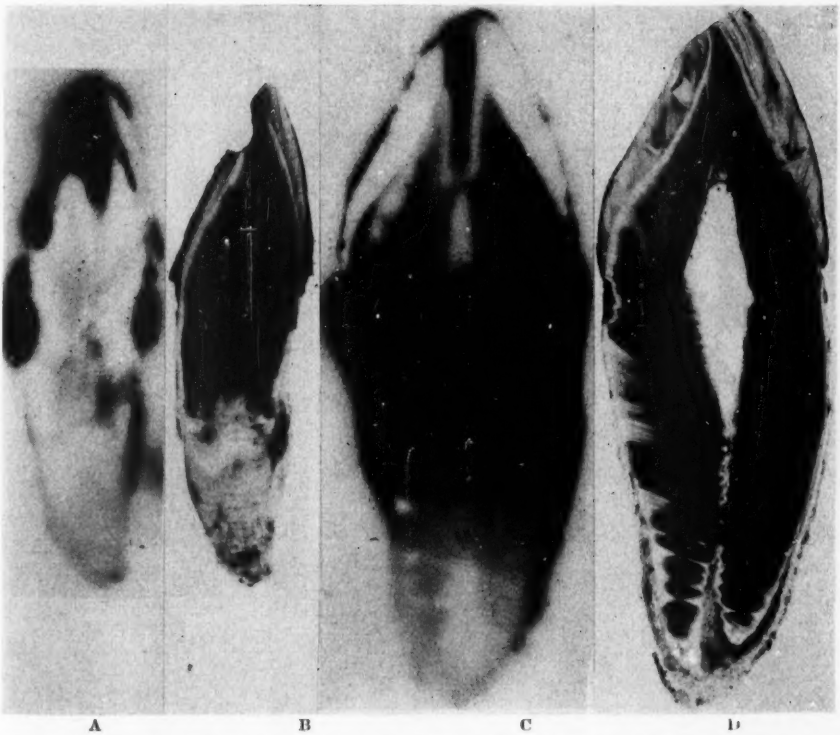
Fig. 3. Radioautographs (equivalent exposures—A: 17 hours, B: 3 days 17 hours) of ground section (C) from upper left second molar extracted after administration of 25 mc. I^{131} to a 63 year old male.

Hence the exposures in this experiment have been corrected and appear as though they were started on the day of extraction and are expressed as "equivalent exposure time."

RESULTS

Fig. 1A is a radioautograph from a mesiodistal section through the lingual half of an upper left second bicuspid of a 33 year old male who was given 25 millicuries (mc.) of I^{131} five hours before the tooth was extracted. The equivalent exposure is 10 days. Note the appreciable amount of iodine deposited in the cellular cementum. This is believed to have come from the periodontal vessels. Most of the iodine deposited in the dentin is assumed to have come from the pulpal vessels. The slight amount on the enamel surface, that from the occlusal groove and that at the cervix are assumed to have come primarily from the saliva and from the original radioactive solution which was taken in a glass of water. Fig. 1C is of a central section from the same tooth showing a similar distribution of iodine. The equivalent exposure is 10 days. Ingress through the carious enamel of the occlusal surface is recorded and a considerable path to the pulp has been penetrated from the linguogingival. Again the cellular cementum of the apical region is permeated.

Fig. 2 shows two sections from the upper left lateral incisor of a 43 year old male who received 25 mc. of I^{131}, 17 hours before the extraction. The transverse section of Fig. 2A and B was taken through the middle of the root while that of Fig. 2C and D was taken near the apex. The equivalent exposure times were 2 days 5 hours and 5 days 10 hours, respectively. Both show localization of iodine in the pulp, in large sections of the dentin, and throughout the cementum. Several segments of dentin appear to be translucent and poorly penetrated by the iodine. The dark areas on



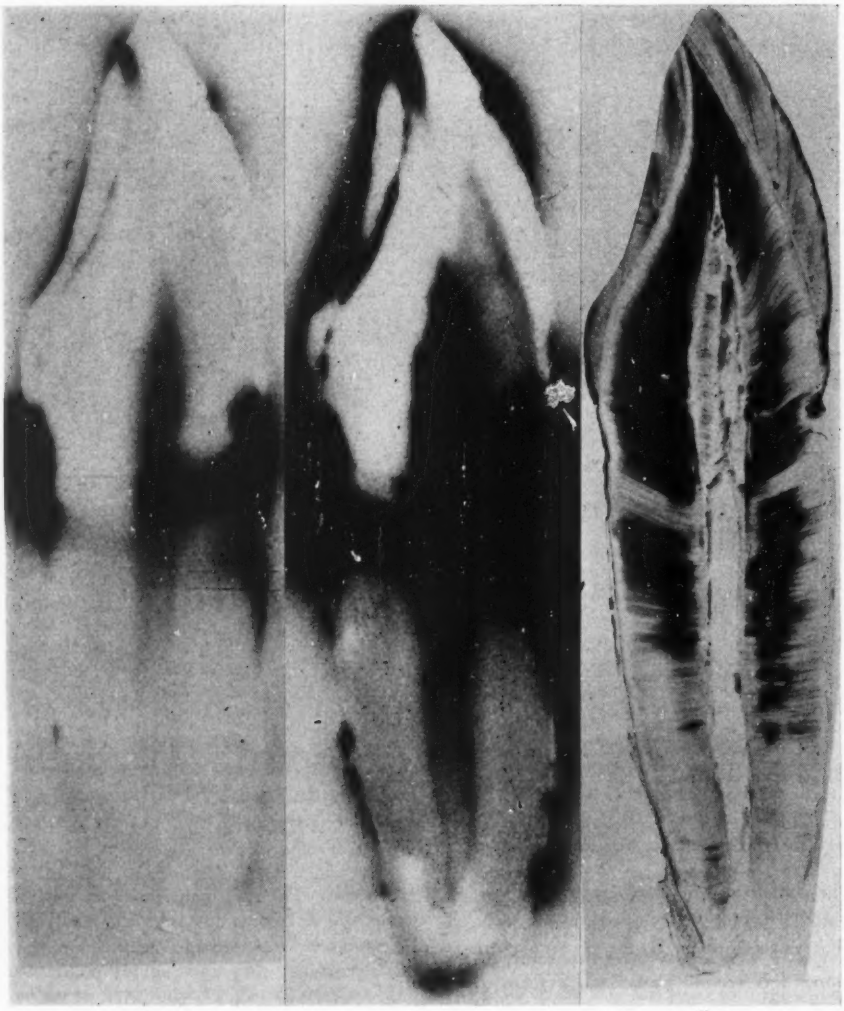
A B C D

Fig. 4. Radioautograph (A) of ground section (B) from lower left central incisor of same patient as Fig. 3 (extracted at the same time). Radioautograph (C) of ground section (D) from upper right cuspid of same patient as Fig. 2 (extracted at the same time).

the cementum are deposits of calculus on exposed root surface.

Fig. 3 shows two radioautographs of an upper left second molar tooth from a 63 year old male, extracted 18 hours after administration of 25 mc. of radioactive iodine. The radioautograph of Fig. 3A was exposed an equivalent of 17 hours. This lighter exposure shows the pathway of penetration at the gingival line on the mesial (right) side. In Fig. 3B, with an equivalent exposure of three days and 17 hours, heavy penetration is seen in the carious area under the occlusal class I amalgam filling, in the pulp, and along the cemental surface. In the photograph of the specimen, Fig. 3C, the amalgam filling has fallen out. The specimen shows secondary caries and discoloration under the amalgam filling, as well as damaged enamel on the mesial surface below the contact point.

Fig. 4A shows the radioautograph of a lower left central incisor from the same patient as Fig. 3. The tooth was extracted at the same time as the previous one. Fig. 4B is a photograph of the section which was made labiolingually through the distal half of the tooth. The radioautograph with an equivalent exposure of 3 days 17 hours shows heavy penetration of the exposed incisal dentin, seepage along the dentoenamel junction of the lingual enamel. The cementum is diffusely penetrated especially in the apical region. Fig. 4C is the radio-



A B C

Fig. 5. Radioautographs (equivalent exposures—A: 17 hours, B: 3 days 17 hours) of ground section (C) from upper right lateral incisor of same patient as Fig. 2 (extracted at the same time).

autograph of an upper right cuspid tooth extracted from the same patient as in Fig. 2 and at the same time. The exposure time was the equivalent of five days ten hours. The cementum is heavily penetrated; the exposed cervical dentin and exposed incisal dentin are grossly penetrated. All of the dentin of the tooth shows some penetration from the pulp chamber. The enamel shows a surface uptake of iodine especially in some areas which appear damaged in the specimen, Fig. 4D.

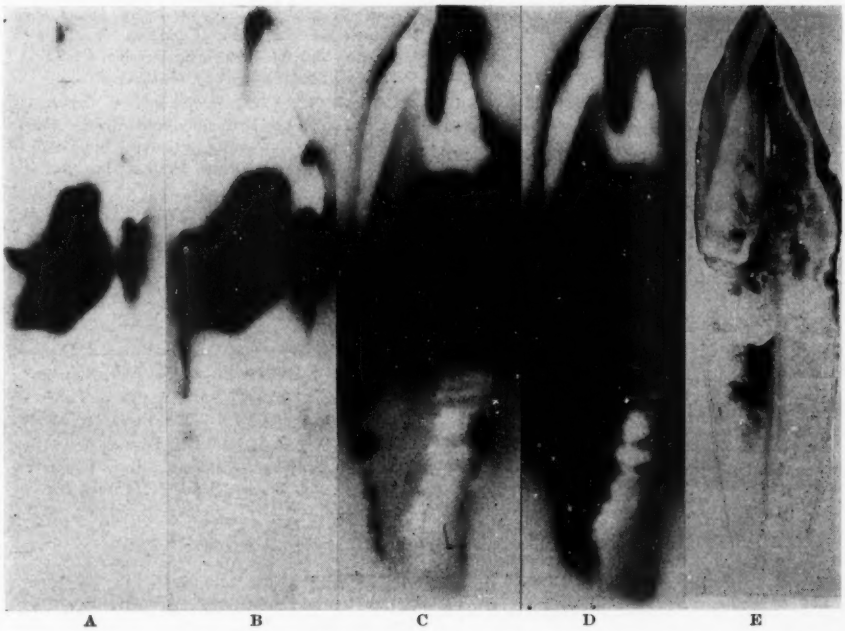


Fig. 6. Radioautographs (equivalent exposures—A: 4 hours, B: 17 hours, C: 3 days 17 hours, D: 13 days) of ground section (E) from the tooth of Fig. 5.

Figs. 5 and 6 show the radioautographs and sections from an upper right lateral incisor extracted from the patient of Fig. 2 and at the same time. Fig. 5C shows a longitudinal labiolingual section through the central portion of the tooth. Fig. 5A is a radioautograph with an equivalent exposure of 17 hours; it shows the detailed uptake of I^{131} at the labiogingival from the surface to the pulp. This corresponds to the translucent zone which extends pulpally from the exposed dentin of the labiogingival area. Fig. 5B is the radioautograph of the same specimen with an equivalent exposure of 3 days 17 hours. It shows the extent of the heavy penetration at the labio- and linguogingival as well as at the incisal dentin and the dentoenamel junction under the lingual enamel plate. The pulpal cavity shows a heavy deposition of iodine which has spread through much of the dentin of the tooth. The cementum surface shows an uptake of iodine in the middle portion of the root while in the apical portion there is a rather wide band of penetration of cellular cementum.

The second section of the tooth of Fig. 5 was made a little to the mesial and is shown in Fig. 6E. The radioautographs of Figs. 6A, B, C, and D were made with equivalent exposures of 4 hours, 17 hours, 3 days 17 hours, and 13 days, respectively. The details of the penetration from the exposed labial dentin through the tooth are seen in Fig. 6A. The longer exposures show surface uptake on the enamel, cementum, and deeply into the exposed incisal dentin.

SUMMARY AND CONCLUSIONS

1. *In vivo* human examinations of cementum metabolism are easily made on

teeth from patients treated with radioactive iodine for thyroid disease.

2. Cellular and acellular cementum were often diffusely penetrated by I^{131}. Complete penetration occurred in teeth exposed to I^{131} *in vivo* in as short a time as 5 hours and was still present in teeth exposed to I^{131} as long as 18 hours. Presumably the time limits are much wider.

3. Exposed cervical dentin was penetrated deeply to the pulp chamber. Deep penetration occurred through secondary dentin as well, confirming the observations of Bartelstone.

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Local Eosinophile Count in Gingival Disease

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HEALY, Daly, and Sweet[1] and Harkavy[2] have sugested that many instances of gingivitis are of an allergic etiology. Healy, et al., reported that simple gingivitis and pregnancy gingivitis were frequently associated with allergic phenomena, such as an elevated gingival eosinophile count (over 4%), and described the successful treatment of gingivitis on an allergic basis. Blood eosinophile counts over 4% are considered outside the normal range[3,4] and when found in cases of suspected allergy are often cited as confirmatory evidence of allergic disease.[5,6] Stine and Burket,[7] however, were unable to confirm the existence of a local (gingival blood) or systemic (peripheral finger-tip blood) eosinophile response in gingival disease but did not include normal controls in their study. The present study** attempts to clarify these contradictory findings.

METHODS

A gingival blood smear was taken from 49 patients, 18 men and 31 women, age 17 to 53, each of whom presented with marked gingivitis. Blood specimens were obtained following gentle scaling of an interdental area of gingival inflammation by sponging up a drop of blood with a cotton pledget. The blood droplet was transferred to a slide, smeared, fixed, and stained with Wright's stain. A differential

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TABLE 1.

Distribution of Patients According to the Gingival Blood Eosinophile Count

% Eosinophiles	Normal Group	Gingivitis Group	Pregnancy Gingivitis Group
0	16	12	5
1	9	15	2
2	11	16	1
3	1	4	2
4	3	1	-
5	-	1	-
6	-	-	-
7	1	-	-
Total Cases	41	49	10
Mean % Eosinophiles	1.3	1.4	1.0

white blood cell count was obtained in terms of the percentage of the various white cells present in each smear. Forty-one patients of similar age and sex distribution without clinical evidence of gingivitis were studied in a like manner and were considered as the control group. Ten patients presenting with pregnancy gingivitis were also observed. No frank manifestations of allergy were observed in any of the 100 patients during the course of this investigation.

Since the studies of Healy, et al. and Stine and Burket,[7] antihistamine drugs have become available. To further study the role of allergy in gingival disease, Pyribenzamine hydrochloride, which has been shown to be efficacious in alleviating symptoms of allergic states, was administered to 15 patients with severe gingivitis. The dosage was 50 mg three times a day for 4 days. Another group of 15 patients with severe gingivitis received a placebo tablet (containing the same ingredients, except for the replacement of the Pyribenzamine by a filler) in the same dosage regimen.

RESULTS

The distribution according to the percent of eosinophiles in the gingival blood smear of the 100 patients is listed in Table 1. There was no marked difference in the eosinophile count among the three groups. In each group the counts were normal in relation to the accepted percentage of eosinophiles usually found in peripheral blood smears (1-4%).[3,4]

The mean percent values of each of the other four principal white cells appearing in the gingival blood smears are presented in Table 2. There are no statistically significant differences between the percentages of white blood cells of any of the three groups.

Following the administration of Pyribenzamine hydrochloride to 15 patients with severe gingivitis, and the administration of placebo tablets to another group of 15 patients with severe gingivitis, no

TABLE 2.

Gingival White Blood Cell Counts in Gingival Diseases

Group	No. of Cases	Neutrophiles* mean	σ	Lymphocytes mean	σ	Monocytes mean	σ	Eosinophiles mean	Basophiles mean
Control	41	65.2	4.5	31.4	5.1	2.2	1.5	1.3	0.17
Gingivitis	49	65.6	7.6	30.8	8.2	1.8	1.7	1.4	0.24
Pregnancy Gingivitis	10	65.6	8.1	30.9	8.8	2.1	1.3	1.0	0.20

*Segmented and non-segmented forms.

Standard deviation (σ) was calculated as $\sqrt{\frac{\xi x^2}{N} - \bar{x}^2}$ where ξ = sum of; x = individual eosinophile counts; N = number of subjects studied; and $\bar{x}$ = mean eosinophile count.

change was noted in the gingival condition of either group, nor was any change apparent in gingival sensitivity or gingival bleeding incident to scaling.

SUMMARY AND CONCLUSION

The white blood cell counts obtained from smears of the gingival blood of 100 patients were determined. Forty-one patients with normal gingivae, 49 patients with marked gingivitis, and 10 patients with pregnancy gingivitis were studied. No difference in the eosinophile count (or other WBC counts) of the gingival blood was noted amongst the three groups. A therapeutic test with a recognized antihistaminic agent (Pyribenzamine) produced no clinical change in the gingival condition of 15 cases with severe gingivitis. These findings suggest that routine cases of gingivitis or pregnancy gingivitis most probably do not have an allergic basis.

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Histologic Topographic Changes of Inflammatory Origin in the Gingival Fibers*

by HENRY M. GOLDMAN, D.M.D., *Boston, Mass.*

ALTHOUGH the clinical appearance of the gingiva in disease state has been described repeatedly, as have the histologic findings, little mention has been made concerning the changes in the pattern of the gingival fiber apparatus.

In a previous report[2] the topography and function of the gingival fibers in health were discussed. It was demonstrated that the gingival fibers consist of coarse collagenous fibers embedded on one end in the cementum (Sharpey's Fibers) and terminating on the other in the papillary layer of the gingiva subjacent to the epithelium. The fiber bundles pass outward from the cementum in groups breaking up in the gingival corium into a meshwork of small bundles, the fibers of which interlace with one another. While the course of the fibers can be determined in sections stained with hematoxylin and eosin, they are best viewed either with Wilder's silver stain or another connective tissue stain.

In the buccolingual aspect, beginning subjacent to the epithelial attachment, connective tissue fibers arising from the cementum pass out at right angles to its surface for a short distance and then bend occlusally, passing into the gingiva and terminating in the papillary layer of the gingival corium. The fibers subjacent to those just described arise from the cementum and run across the gingiva at right angles to the tooth, becoming incorporated in the papillary layer of the

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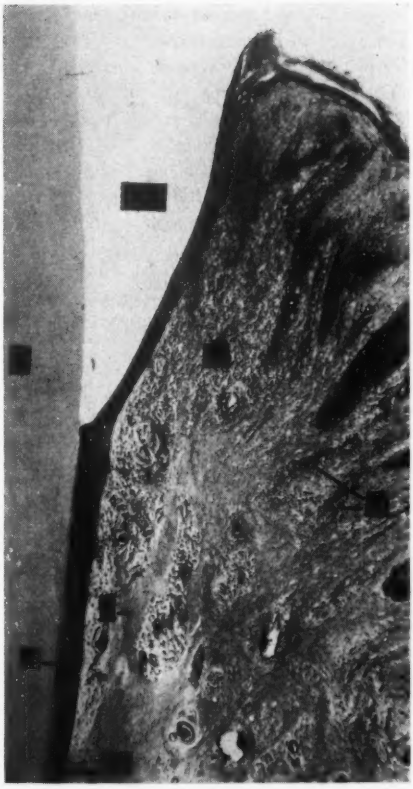


1. ES, enamel space; D, dentine; C, cementum; EA, epithelial attachment; IN, inflammation; GF, gingival fibers; A, remnant of gingival fiber; B, inflammatory cells interspersed between gingival fibers.

gingiva. A little farther apically the fibers arise from the cementum, pass at right angles to the tooth directly over the alveolar crest, and incline apically between the outer periosteum of the alveolar process and the epithelial covering of the attached gingiva. These latter fibers join with small fibers arising from the outer periosteum and on the other side with fibers running into the papillary layer of the corium These bundles form a distinct group tending to attach the tooth to the outer layer of the periosteum and also to keep the attached gingiva closely adapted to the underlying bone and tooth.

On the proximal sides, the connective tissue fibers arise from the cementum at the same level as those on the buccal and lingual aspects. The group subjacent to the epithelial attachment passes occlusally, travelling parallel to the crevicular epithelium, and terminates in the papillary layer. The next adjacent group of fibers also passes occlusally and seemingly makes up the framework for the interproximal gingiva. Directly subjacent to these, the fibers travel across the interproximal space over the alveolar crest and are attached to the adjacent tooth; thus these bundles form a distinct group which binds one tooth to another (the transseptal fibers). When labiolingual sections directly above the alveolar crest are studied, cross sections of these fibers are apparent.

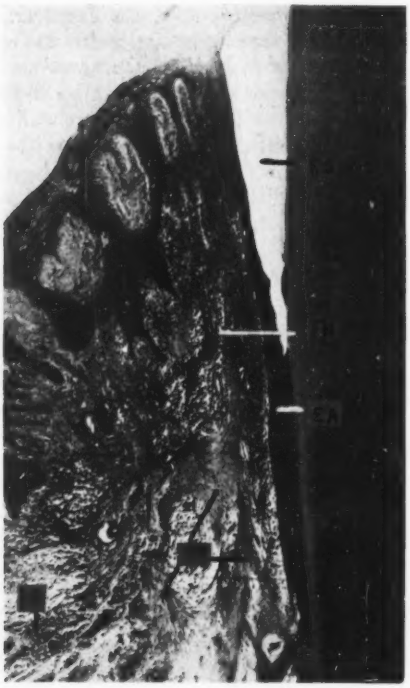
The purpose of this report is to demonstrate the histologic and topographic changes in these fibers in gingival inflammation in cases where no clinical recession is present.

MATERIAL AND METHODS

The present study is based upon the microscopic examination of sections of three human jaws; the ages were 22, 32, and 48. These sections were selected because neither clinical inflammatory hyperplasia of the gingiva nor recession was present, although the gingivae showed distinct inflammatory changes: minor changes in color and consistency, areas of retraction of the gingival tissue from the tooth, shallow pocket formation associated with calculus deposits. The sections studied were taken from various regions of the maxilla and mandible. The microscopic sections were prepared by the celloidin method and stained with hematoxylin and eosin; some sections were stained with Wilder's silver stain.

FINDINGS

Microscopic examination disclosed varying degrees of inflammatory infiltration in



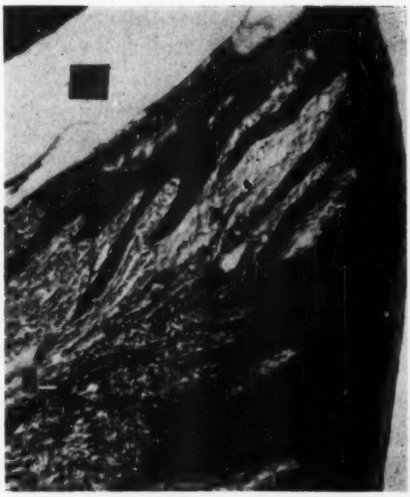
2. ES, enamel space; IN, inflammation; EA, epithelial attachment; C, cementum; GF, gingival fibers.

the gingival tissue and also accompanying changes in the architectural pattern of the gingival fibers. To demonstrate these changes six photomicrographs are presented.

Fig. 1 is a photomicrograph of a buccolingual section illustrating the changes seen in the gingival fiber apparatus. The gingival margin lies on the enamel surface, the epithelial attachment running from almost the gingival margin onto the cemental surface of the tooth. The sulcus is practically absent. (Ulcer formation at the gingival margin adjacent to the tooth surface was seen in other sections in the serial sections studied.) Hornification of the epithelium is apparent. The group of fibers which usually run parallel to the crevicular epithelium is replaced by a dense inflammatory infiltrate; remnants of a subjacent group, however, can be discerned at "A." The lateral group of gingival fibers are still relatively intact, although in many areas "B," inflammatory cells are interspersed between the collagen bundles. Nevertheless, the original topography can be recognized as evidenced by groups of fibers passing laterally and ending in the papillary layer of the surface epithelium. The apical portion of the epithelial attachment can be seen in apposition to a group of fibers which pass occlusally and are parallel to the epithelial attachment.

Fig. 2 is a photomicrograph of a buccolingual section demonstrating a more advanced change in the pattern of the gingival fibers. The gingival margin as in the first section is on the enamel surface and the epithelial attachment, here too, extends from almost the gingival margin onto the cementum of the root. Thus, the sulcus is practically non-existent. The fiber apparatus, however, is distinctly broken up and the inflammatory infiltrate is more diffusely spread through the gingival tissue. Only a remnant of the original fiber apparatus can be discerned, the collagen fibers being almost entirely replaced by inflammatory cells. Remnants of the fiber bundles can be seen at "GF." Of interest is the dense inflammatory infiltrate adjacent to the apical portion of the epithelial attachment which lies in apposition to a bundle of still attached fibers. While the outer border of the epithelium is distinctly hornified, a proliferation of the epithelium into the corium is seen which can be interpreted to be of inflammatory origin.

Fig. 3 is a photomicrograph of the gingival tissue at the marginal area (Wilder's silver stain); the gingival margin lies on the enamel surface of the tooth. The epithelium is hyperplastic and there is marked elongation of the rete pegs. Only a few of the coarse, broad fibers "F" which usually compose the gingival mat can be seen. The terminal endings in the papillary layer are

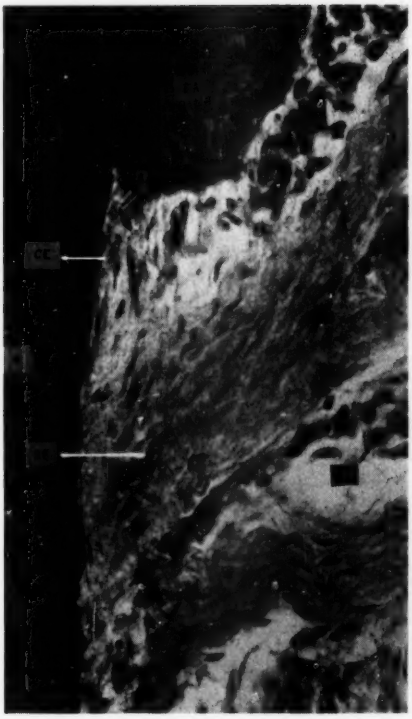


3. ES, enamel space; E, epithelium; F, remaining coarse fiber stained with Wilder's silver stain.

almost completely absent as well as the normal criss-cross pattern of the fibers.

Fig. 4 is a photomicrograph of the epithelial attachment alongside the cementum of a tooth and the subjacent tissue of the gingiva. The apical proliferation of the epithelial attachment "EA" is seemingly limited at "A" by a degenerated remnant of connective tissue fibers. A moderately dense inflammatory infiltrate lies adjacent to the epithelial attachment, "IN"; also the inflammatory reaction is present more deeply in between fiber bundles, "IN". More apically, gingival fibers are attached to the cementum and a cementoblastic layer is present. However, the connective tissue fibers appear blotchy in nature having fibrinoid quality.

Fig. 5 is photomicrograph of an area of fiber bundles subjacent to the gingival epithelium. The coarse fibers are interspersed with inflammatory cells chiefly plasma cells and lymphocytes. Edema is present between the fiber bundles and the blotchy appearance of the individual fiber is apparent.

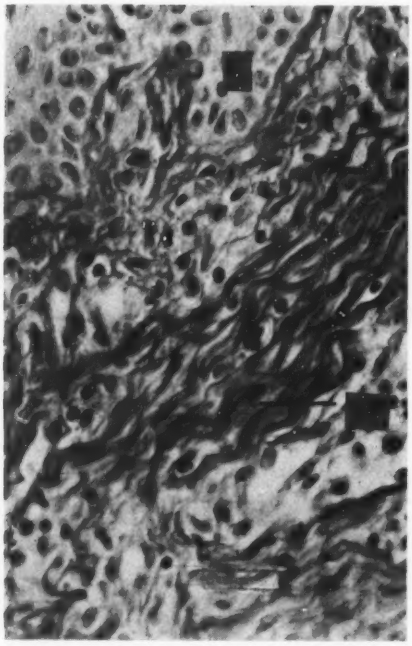


4. C, cementum; EA, epithelial attachment; IN, inflammation; CE, cementoblastic layer; GF, gingival fibers; A, degenerated remnant of connective tissue fibers.

DISCUSSION

The microscopic study of these selected sections disclosed a chronic inflammatory process in the gingiva, progressive in nature, destroying the gingival fiber apparatus. The inflammatory infiltrate is seen interspersed between fiber groups and later fiber bundles. Areas first destroyed are the midsections of the fiber bundles, the cemental portions of the fiber groups remaining intact for a subsequent period of time prior to complete loss.

The early manifestations in the gingival fiber apparatus due to chronic gingival inflammation are characterized by the de-



5. E, epithelium; GF, gingival fibers.

struction in the middle sector of those fibers which originate directly subjacent to the epithelial attachment (described as the first group of the gingival fiber apparatus). The area is infiltrated chiefly by plasma cells, lymphocytes, and histiocytes. These inflammatory cells intersperse themselves between fiber bundles, resulting in blotchy appearance of the connective tissue fiber. Later changes are characterized by a dense inflammatory infiltration of the gingival corium progressively replacing the connective tissue gingival fibers. Perivascular infiltration of inflammatory cells is a conspicuous feature. Occasionally there is a proliferation of endothelial cells lining the blood vessels and a partial or complete occlusion of the vessel lumen may be noted. Varying degrees of fibroblastic proliferation, a feature of the repair process, were noted.

In some sections studies (not illustrated), this repair process was marked (evidenced by inflammatory gingival hyperplasia). It is to be noted, however, that this proliferation of connective tissue does not simulate the architecture of the previous existing gingival fibers, but is present in a random fashion dictated by the inflammatory process.

SUMMARY

Cases of marginal gingivitis were studied. These had no clinical recession or distinct hyperplasia. Alterations in the gingival fiber topography and histology were described as a progressive destruction due to inflammation. In the late stages, the gingival fiber apparatus may be completely replaced inflammatory elements. Varying degrees of repair were seen.

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Case Report

A 76-Year Old Man with Healthy Periodontium

by IRWIN WALTER SCOPP, B.S., D.D.S.,* *New York, N. Y.*

DENTAL literature is replete with case reports and papers on diseased periodontium. Searching inquiries abound in the etiology of periodontal disease. It is comparatively rare to read a case report discussing a healthy mouth, periodontally speaking, that has withstood the test of time.

Even textbooks on periodontia give scant consideration to healthy supporting structures. When they do consider healthy periodontium, generally photographs in color will be shown of a 21-year old female, very appealing dentally and otherwise. In viewing these descriptions of the normal mouth, one wonders about the condition of this girl's teeth when she reaches the age of fifty or sixty or perhaps even seventy.

The case illustrated is that of a 76-year old Spanish American War veteran with healthy periodontal structures. This veteran states that twelve years ago he had coronary thrombosis and as a result gave up working at his occupation of trumpet player in a popular orchestra. Later, he became a band leader and played the violin and banjo in addition to the trumpet. Throughout his adult life, he earned his livelihood traveling around the country playing the trumpet in night clubs and as a vaudeville entertainer. It can well be imagined that this itinerant mode of life was not conducive to good diet or general health. For at least forty years, during his adulthood, he consumed one pint of whiskey daily. After his coronary attack, however, he abstained from alcohol. Since the age of sixteen, he has been a habitual smoker. He does not smoke cigarettes, but often smokes cigars when he is outdoors and pipes when indoors. This has been practiced to the extent of six to ten cigars per day, and approximately fifteen pipefuls per day and he has continued this up to the present time.

He is the youngest of thirteen siblings, all born in Virginia. He states that all his brothers and sisters were edentulous and wore replacements at an early age. His parents, too, were edentulous at the time of their death.

Physical examination revealed a well developed, colored male, 5 ft. 6 in. in height, weighing 147 lbs. His blood pressure was $^{130}/_{80}$. For the past seven years, he has had a bilateral hernia. His blood, urine, electrocardiograph and fluorscopic examinations were all within normal limits. X-rays of his chest show the aortic arch as elongated and configuration of the aorta as compatible with hypertensive or arteriosclerotic disease. There is no history of anginal pains or objective symptoms of hypertensive disease.

About forty-five years ago, following a genito-urinary infection, probably of gonococcal origin, he developed a stricture of the urethra whcih is presently manifested by a small split stream. There is no history of Lues. He started wearing bifocals about eleven years ago.

Continuously throughout his life, in brushing his teeth, this veteran used salt on a brush. His brushing was of a haphazard nature, sometimes using a push-pull, sometimes a rotary movement, but never any of the more accepted toothbrush methods employing pressure and release.

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"Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration."

Figure I. Note accumulation or sordes, bacterial slime and calculus around all the teeth. The gingiva are highly keratinized, dense and massive. Architectural form is excellent. The end to end bite of the anteriors and the close interdigitation of the upper and lower teeth are evident. The upper central incisors show cracks which have been stained by excessive use of cigars and pipes.

He would brush his teeth once a day.

He visited dentists on three or possibly four occasions. At these times, he received an alloy filling and a prophylaxis. He states that his teeth never caused him any trouble.

Photographs reveal well developed, stippled, firm gingiva. Interproximally and around necks of the teeth, they end gracefully and are knife-edged. They appear highly keratinized, thick and very dense. Despite vigorous probing, there was no bleeding. The gingival crevice is knife-edged with the crevice depth of about 1 mm. and the gingival architecture approaches the ideal. In lateral excursion and in protrusive, the teeth glided from side to side with such precision and beauty as to compare with the gears of an intricate, precision-cut mechanism. There was no mobility of the teeth. Calculus, bacterial slime, and sordes were plentiful.

The incisal and occlusal surfaces of the teeth are severely abraded and stained with tobacco and food. The upper left third molar has a small alloy occlusal filling. The lower left second bicuspid has a distal occlusal filling, and the lower right second molar has a mesial occlusal gold inlay. On the upper right first molar, due to abrasion, the occlusal surface is cupped out, but no caries is present.

Labially on the upper central incisors and lingually on all the upper anteriors, there are hair-like cracks in the enamel. These minute faults are accentuated by infiltrations of tobacco stains. The photographs show this clearly.

Between the lower left cuspid and lower left first bicuspid, there is a space which normally could be considered ideal for food impaction. Further, the upper left cuspid serves as a plunger cusp forcing food into the lower gingiva. The space is such that food can be trapped easily in this area. Similarly, a plunger cusp action was found between the upper left cuspid and the upper left first bicuspid, with the lower left first bicuspid directing the plunger action. Clinically, examination of the gingiva reveals no sign of irritation or inflammation. Radiographs show no loss of alveolar process in these areas. The teeth are firm.

Figure II. The lower teeth show severe abrasion resulting in short clinical crowns. The abraded areas are heavily stained by the excessive use of tobacco. The abundance of calculus, bacterial slime and sordes is evident. The dense, highly keratinized gingiva with a favorable gingival architecture is shown. The gingiva hug the teeth tightly with no gingival crevice exceeding 1 mm.

DISCUSSION

It would be illogical and, indeed, indiscreet to draw conclusions from one case. Yet such cases are so extremely rare as to negate group studies. Reporting of this one case may bring to mind others that have been observed by practicing periodontists. It may be of interest and perhaps revealing to view this case in the light of recent research.

Waugh, McCarrison and Price found healthy periodontium in tribes having primitive protective diets. Waugh reported that the Eskimo of Labrador and Alaska were free from periodontal disease by virtue

of their diet consisting of seal meat, fish, and native berries. The teeth were worn down almost to the level of the gingiva at the age of forty, an advanced age for Eskimos. The shortening of the clinical crown contributed to the healthy periodontal condition.

Price, in his field studies through Alaska, Australia, New Zealand, South Sea Islands and Africa, made similar observations. In considering these studies, one wonders how this 76-year old man, who has subsisted on an American diet, has thirty-two teeth in his mouth and a healthy periodontal structure.

Gottlieb, in 1921, reported changes in the supporting structures from youth to old age. He formulated five postulates on the physiology of teeth and the periodontium. As listed below, these comprise the normal evolution of teeth: (1) migration of the epithelial attachment towards the apex (2) the simultaneous atrophy of alveolar crests (3) the protrusion of the teeth towards the occlusal line (4) the mesial displacement of teeth (5) the compensative abrasion of the teeth, both at the occlusal surface and at the points of contact. In reviewing this case against these postulates, the abrasion of the teeth is the sole point upon which this man's dentition complies with the above at the age of seventy-six.

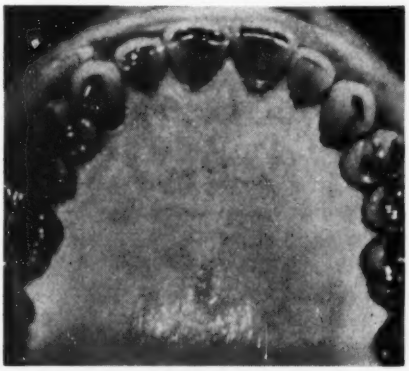

Figure III. Note the well developed symmetrical arch. The palate and palatal gingiva are thick, dense and highly keratinized. Abrasion is very pronounced with tobacco stains evident on all teeth. On the anteriors, there are minute cracks in the enamel which have become stained with tobacco.

Merritt and Hartzell believe oral sepsis to have a major role in the etiology of periodontal disease. In this particular mouth, despite the abundance of sordes, bacterial slime, calculus and all the bacteria encouraged by such a condition, the epithelial attachment held firmly, the alveolar bone remained well formed and the gingiva resistant and healthy.

Boyle, Wolbach & Howe produced vitamin C deficiency in experimental animals and caused periodontitis complex. Mellanby showed that periodontal disease in dogs can be produced and prevented by variation in vitamins A and D. Boyle and Bessey reported bone resorptions from lack of vitamin A in the diet. Sinclair showed that multiple vitamin deficiency could cause periodontal disease. In considering this case in the light of the above research, it becomes clear that in this particular man, where lack of adequate diet and vitamins existed, no detrimental effects on periodontium were noted.

McCollum, McLean and Granger do mouth rehabilitation to obtain ideal articulation to correct eccentric intercuspation. If the bite of this patient had been altered by reconstruction, perhaps at the age of fifty, one wonders what the result would be at his attained age of seventy-six.

Glickman, at the 1951 Meeting of the Academy of Periodontology and in his many previous publications, stressed the importance of systemic factors in periodontal disease as always being present, although as yet many of them are unknown. These factors strongly influence periodontal health or disease. Local factors which will cause rampant periodontal disease in one mouth will produce no derogatory effects in another. In considering this case, one can readily understand that although local factors encouraging periodontal breakdown

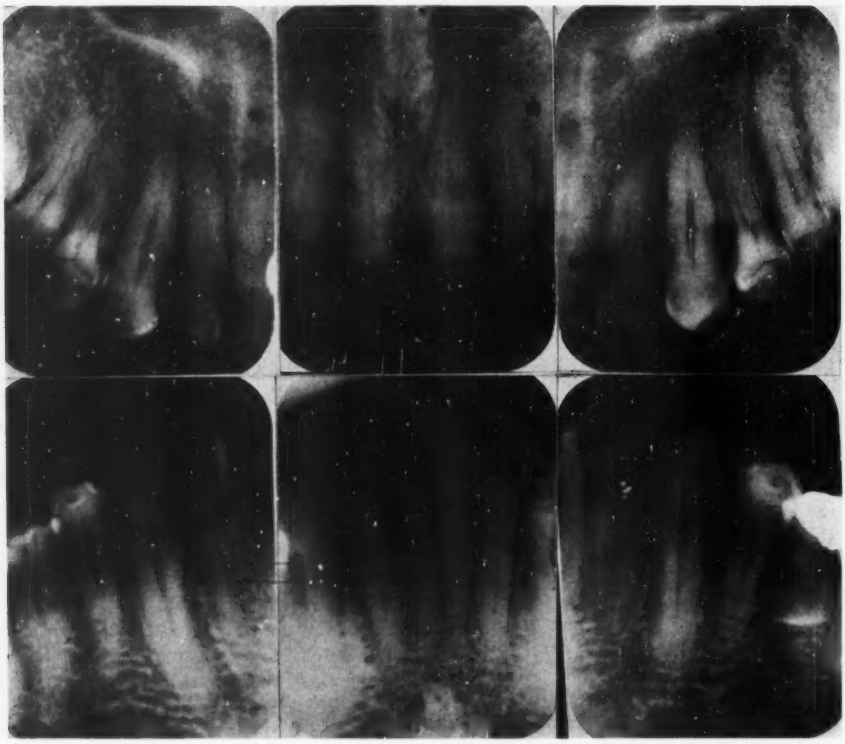

Figure IV. The lamellae are distributed in an orderly manner showing the density of the bone. The lamina dura is continuous and intact. The amount and distribution of the bone is favorable.

were present, something in the systemic make-up of this man rendered him immune to any periodontal disease. In viewing this case, one becomes convinced that systemic factors, although unknown, play a very, very, important role in maintaining health. As yet, uncovered knowledge in the biochemistry of tissues will eventually shed light on this phenomenon.

The biochemistry and physiology of fat soluble steroid compounds found in the gonads and adrenal cortex are intimately associated with bone physiology. Fuller Albright discusses three factors which influence osteoblastic activity: (1) steroid hormones (2) mechanical stress and strain (periodontists deal with this daily) (3) nitrogenous building blocks.

In considering steroids as they affect the bone matrix, Albright divides them into two groups: anabolic, those which stimulate; and antianabiolic, those which inhibit bone activity. The anabolic steroids are estrogens and androgens and are derived from the ovaries, testes and adrenal cortex. The antianabolic steroids which inhibit bone formation come from the adrenal cortex, and are called the sugar or "S" hormones.

After menopause, or the male climacteric, osteoporosis generally occurs. This may be explained as the loss of bone stimulating effect of the estrogens and androgens. Conversely, the persistence in activity of anabolic steroids of estrogens and androgens may continue to a great extent the osteo-

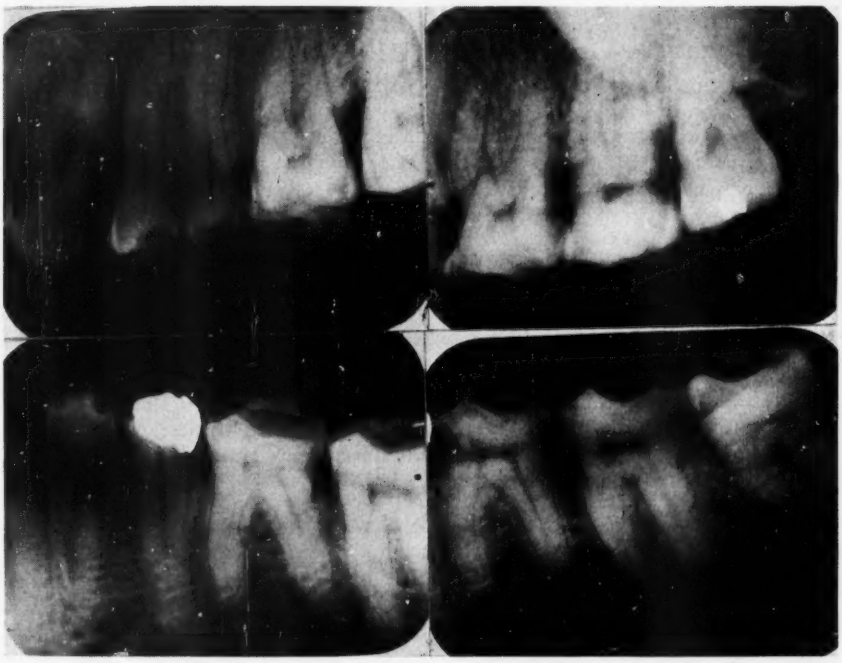

Figure V same as Figure IV.

blastic activity.

The heavy bone formation in this patient may be explained on the basis of anabolic steroids. Further, the high keratization or hornification of the gingiva in this case may be explained also on the basis of steroid hormones. Ziskin has demonstrated the effect of hormones on the hornification of gingiva.

As stated above, no conclusions should be drawn from observations and the study of one case. However, the paucity of knowledge on the etiology of periodontal disease should be an incentive for further study and research.

(From Veterans Administration, 252 7th Ave., New York 1, N. Y.)

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Editorial

Scaling Teeth Versus Subgingival Curettage

There would seem to be some confusion regarding the correct use of the terms "scaling" and "subgingival curettage," as frequently noted in describing these operations. They are often used synonymously. Accurately speaking this is incorrect.

Scaling is defined as "the removal of scale-like deposits of calculus from the crowns and roots of teeth."[1] "The operation of removing deposits of salivary or serumal calculus from the teeth and their necks."[2] The act of removing calculus from the teeth."[3] "An operation which consists in the removal of deposits of calculus from the teeth."[4]

Curettage, on the other hand, is defined as "gently scraping infectious material from the root surfaces of the teeth where the investing tissues are detached."[2] "Removing diseased tissues or accumulated secretions from the cavity or surface."[1] "Scraping away exuberant or dead tissue."[4]

From the above definitions, it will be obvious that the terms "scaling" and "curettage" cannot be used interchangeably. Scaling teeth, as generally used, refers to the removal of supragingival calculus. While in some cases of periodontitis, there are deposits of so-called serumal calculus and scaling may be necessary, such deposits are, as a rule, relatively insignificant and their removal a minor part of the operative procedure.

Too often, however, the operation of subgingival treatment is referred to as "scaling teeth" creating the impression that the operation consists of the removal of calculus from the root surfaces. This is misleading. The operation is essentially that of removing "infectious material from the roots of teeth," leaving a smooth and polished surface. Since this is true, a more accurate description of the operation would be that of curettage. It also has the added advantage of being widely known in scientific circles and should be employed as the correct term in the subgingival treatment of periodontal pockets.

The use of such terms as "apoxesis" and "odontexsis" which may be etymologically correct, are not in general use and serve to add to the confusion. They might well be omitted from the nomenclature of periodontology.

A. H. M.

1. Dunning & Davenport. A Dictionary of Dental Science and Art.
2. Ottofy. Standard Dental Dictionary.
3. Anthony. Dental Dictionary.
4. Blakiston's New Gould Medical Dictionary.

DR. JAMES ALEXANDER SINCLAIR

1877-1952

On March 15, 1952, while on a fishing trip near his home in Asheville, North Carolina, Dr. James Alexander Sinclair suffered a heart attack and died before reaching his home.

Dr. Sinclair was born December 8, 1877, the son of Col. & Mrs. Peter J. Sinclair. He attended the North Carolina State College at Raleigh and was graduated in 1900 from the Baltimore College of Dental Surgery and began practice in Asheville in 1902.

Throughout his professional life he was an active worker in his profession—a member of many organizations, including the North Carolina State Dental Association, the Southern Academy of Periodontology (of both of which he was a past president) the American Dental Association, the American Academy of Periodontology, Psi Omega and Omicron Kappa Upsilon dental fraternities, and in 1949 was elected a Fellow of the American Academy of Periodontology.

Dr. Sinclair is survived by his wife, a daughter, two grandchildren, two brothers and four sisters. He will be sorely missed by all who knew him, especially his coworkers. He was tireless in his professional activities, a regular attendant at its meetings and a life-long student in several fields of research. He was one who would have been a credit to any profession. He will be long remembered by all who knew him, not only for his many contributions to his profession but for all that is highest and best in life—unselfish service in the interest of his fellowmen.

A. H. M.

Announcements

Academy Seminar April 28 to 30, in Ann Arbor, Mich.

The annual spring seminar of the American Academy of Periodontology will be held this year in Ann Arbor, Michigan, on April 28, 29, and 30, 1952. All members of the Academy are eligible to attend, and the arrangement committee feels that the program should justify a good attendance. The fee for the course is $25.00 and payable with this application, or at the time of registration. Those planning to attend this meeting should write to Dr. William R. Mann, W. K. Kellogg Foundation Institute, Graduate and Post-graduate Dentistry, Ann Arbor, Michigan. The applicant must also write to the Michigan Union, Ann Arbor, Michigan, and make a room reservation, giving the time of arrival and departure, and mentioning that he is planning to attend the seminar of the American Academy of Periodontology. Guests may attend the meeting if sponsored by a member of the Academy. Each member is entitled to one guest.

The program is as follows:

Monday, April 28, 1952

8:30 Registration—Room 2000 Kellogg Foundation Institute

9:30 "Clinical Implications of Man's Response to Stress"
Dr. Jerome W. Conn, Professor of Internal Medicine, University of Michigan

11:30 Discussion of Dr. Conn's presentation

LUNCHEON

1:30 "Studies on the Physical Properties and Deformation of Bone"
Dr. F. Gaynor Evans, Associate Professor of Anatomy, Wayne University

3:00 "The Effects of Reduced Blood Supply on Bone, Including Density Deformations with Use of Radio-Active Strontium—90"
Dr. Carl C. Coolbaugh, Department of Anatomy, Wayne University

Tuesday, April 29, 1952

9:00 - 12:00 and 2:00 - 4:30—"Traumatic Occlusion—Its Diagnosis and Treatment"
Dr. Donald A. Kerr, Professor of Dentistry
Dr. Sigurd Ramfjord, Ass't. Professor in Dentistry, University of Michigan
Dr. C. Mark Gilson, Instructor in Dentistry, University of Michigan

6:00 Cocktails and Dinner at the Michigan Union

Wednesday, April 30, 1952

9:00 "The Dentist is a Physician; An Exercise in Physical Diagnosis"
2 S.W. Sunporch; University Hospital
Dr. William H. Beierwaltes, Ass't Professor of Internal Medicine, University of Michigan

LUNCHEON

2:00 "The Role of Microorganisms in Periodontal Disease"
Dr. Theodor Rosebury, Professor of Bacteriology, Washington University

4:00 FINIS

The program has been arranged by Dr. Edgar Jones, Detroit, Michigan.

ANNUAL MEETING OF SOUTHERN ACADEMY OF PERIODONTOLOGY

The 32nd annual meeting of the Southern Academy of Periodontology will be held at the Grove Park Inn, Asheville, North Carolina, on May 25, 26 and 27. The officers are: Dr. H. Roger Turner, President; Dr. S. P. Gay, President-elect, and Dr. Earnest L. Banks, Secretary-Treas.

Dr. W. G. McIntosh of Toronto, and Dr. Donald K. Kerr, of the University of Michigan, will be the principal clinicians.

The meeting is open to guests on payment of a $10.00 registration fee. For further information, contact Dr. Leonard S. Turner, Attendance Chairman, 1274 West Flagler St., Miami, Florida.

AMERICAN BOARD OF ORAL PATHOLOGY TO HOLD EXAMINATION

The American Board of Oral Pathology will conduct its next examination in St. Louis, Missouri, on September 7, 1952, the Sunday immediately preceding the opening of the American Dental Association Annual Meeting.

Those who wish to have their credentials evaluated in anticipation of appearing before the examining committee should contact Dr. Joseph L. Bernier, Colonel DC, USA, Secretary-Treasurer, at the Armed Forces Institute of Pathology, 7th Street and Independence Ave., S. W., Washington 25, D. C., as soon as possible.

The necessary application blanks can be secured from Dr. Bernier's office.

THE COLORADO DENTAL FOUNDATION

announces its Sixth Annual Seminar, to be held in the Colorado Springs Fine Arts Center, June 15-19, 1952.

The program will present recent developments in the fields of Anesthesia, Endocrinology and Nutrition; the essayists are to be Doctors Harry Sicher, Hugo Rony, Max Sadove, and Sam Dreizen.

For further information write Dr. Beryl Ritchey, Secretary, The Colorado Dental Foundation, Colorado Springs, Colorado.

Gingival Hyperplasia in Dilantin Sodium Therapy*

By PAUL E. JURGENS, D.D.S., *St. Louis, Missouri*

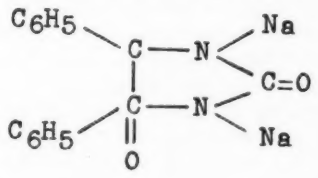
IN 1937, Merritt and Putnam,[21] discovered that dilantin sodium was successful in controlling electrically induced convulsions in animals. By 1939 this drug proved its efficacy on humans in epilepsy. Through the following years it has been successful where other drugs failed, and the best epileptic drug in use.

This drug, an active anticonvulsant, is superior to the bromides and phenobarbital without the undesirable hypnotic effect of either.[16,27,33] After its administration the frequency and number of convulsive seizures are greatly reduced.[13,14] There is also a great improvement in general health, mental attitude, and personality. Sodium dilantin is more successful in treating grand mal than petit mal.[6,21,27]

CHEMISTRY

Sodium dilantin, or diphenylhydantoin sodium, sodium diphenyl hydantoinate, phenytoin sodium, 5-diphenyl glycourea, epanutin (Great Britain), is a derivate of glycolyl urea, and is an odorless, white powder with a bitter taste. It is soluble in water and alcohol, insoluble in benzene and ether, and has a pH of 11.7.[2,3,14]

The formula of dilantin sodium is:[10]



DOSAGE

The correct dosage differs for every individual and is determined by trial.[27] The ideal dose is that amount that will just control the convulsions and will still be below the level of toxicity.[2] It is best administered in capsules.[3,14] The prescribed dosage varies from 1½-7½ grains/day (.2-.6 grams), averaging 4½ grains/day.[18,21,31,32]

TOXIC MANIFESTATIONS

Toxic manifestations that infrequently occur with use of the drug are nausea, dizziness, vertico, ataxia, nystagmus, diplopia, gastric distress, dermatitis, itching of the skin, and hypertrophy of the gingiva.[9,14,15,18,27] The most serious reaction seems to be the gingival hypertrophy.

The gingival changes have been the only hyperplastic one demonstrated to date.[3] Statistical reports since 1939 show variable prevalence, ranging from 3 to 62%.

Author	*Number cases*	*% showing change*
Berg and Perlman[3]	14	57
Esterburg and White[10]	—	54
Fetterman[11]	28	25
Frankel[12]	48	62
Glickman and Lavitus[14]	55	18
Kimball[17]	152	57
Merritt and Putnam[22]	332	3
Millhon and Osterburg[23]	17	35
Pinsky[25]	—	60
Stern and Eisenbud[32]	50	52
Swinehart[34]	—	50
Ziskin, Stowe, and Zegarelli[34]	55	18

The disparity in incidence may be because: (1) the statistics were compiled largely by internists rather than dentists and (2) observations in no instance considered the period of time of administration of dilantin.[32] The usual feeling is incidence in less than half of the cases.

There is no relationship between gingival involvements and sex, age, height, and weight of the patient.[5,7,15,25,34]

CLINICAL PATHOLOGY

The gingival changes may occur as early as two weeks, during dilantin therapy, but

*This paper won first prize in the American Academy of Periodontology's student essay prize contest in 1950-51.

usually it is a matter of months.[25,30,32] The primary lesion is almost invariably without subjective symptoms.[5] Tenderness and hemorrhage are rare;[10,20,34] occasionally the esthetics and difficulty in masticatory function are the primary complaint.[25]

The primary lesion is a painless hyperplasia of the gingivae which starts with an enlargement of the interdental papillae, spreading finally labially and lingually in a semilunar distribution circumscribing the necks of the individual teeth.[14,35] At first it begins as increased stippling and a granular, warty appearance of the gingivomucosal surface. This is succeeded by lobulation, with cleft formation at the coalescence of the lobules. Local inflammatory involvements are uncommon because of a thickened keratinized surface present.[5] When uncomplicated, the gingiva is firm, resilient, pink or pale pink, and has a normal level attachment.[10,14,29,38] Many patients are unaware of the change taking place.[11]

The lesion caused by dilantin sodium is non-inflammatory in nature but local irritants may lead to inflammatory changes. Inadequate oral hygiene results in venous stasis, accompanied by bleeding into tissues and secondary pigmentation, causing a light purple to dusky blue pigmentation.[14] Poor oral hygiene, traumatic occlusion, caries, calculus, and poor fitting crowns aggravate the condition.[10,14,32,37] The removal of irritants serves only to diminish edema and discoloration. In mouths with heavy calculus deposition the hyperplasia may not be present, and conversely, clean mouths may have profuse lesions.[14]

The size of the lesion varies in different mouths and different parts of the same mouth.[5,34] The regions most susceptible to hyperplasia are in the gingival tissues about the labial surfaces of both upper and lower anterior teeth, but particularly in the lower jaw.[10,14,23] When the teeth are separated the lesion formed is smaller because of self-cleansing action.[14,27]

Edentulous mouths or edentulous areas do not develop the lesion.[32,34] The lesion tends to form in relation to the supporting structures of the teeth *in situ*. Hyperplasia will develop around abutment teeth of a fixed bridge; however, it does not develop around the pontics.[3,14]

Tooth irregularity may be sufficient to encourage development of gingival hyperplasia where the basic tendency exists, but are not of themselves the mechanism for the initiation of the lesion.[7,14]

In exaggerated cases of hyperplasia the teeth may drift from pressure from the overgrowth of tissue. Berg and Perlman[3] reported a case of labial drift, due to tissue overgrowth. Swinehart[34] reported an exaggerated case where the teeth were moved into malocclusion by the irresistible expansive force of the proliferating tissue.

HISTOPATHOLOGY

The stratified squamous epithelial layer is fairly thick with few or no mitotic figures.[10] The cells are covered by a keratinized layer.[28] Considerable downgrowth of basal layer epithelium takes place in long strands which break off forming rete pegs and epithelial pearls.[5,10,29,35] In the corium an increase in size and number of collagen bundles takes place with an increase in adult and young connective tissue cells, blood vessels, and intercellular elements.[5,10,14,36] Thoma[36] and Zegarelli and Ziskin[38] have reported cases with bone heteroplasia taking place. Round cell infiltration of lymphocytes and plasma cells are seen in most sections.[10,14] Thoma[35] believes this is a secondary manifestation. From the above picture these changes indicate that a hyperplasia and a hypertrophy both take place. Esterburg and White[10] transplanted some of the tissues directly into the anterior chamber of guinea pig eyes where it could be watched. They concluded the tissue was not malignant.

PATHOGENSIS

The exact cause of the hyperplasia, the disposal of dilantin in the body, and the

method by which dilantin exerts its action is unknown.[2,5,25] The reaction is of a toxic nature and the toxic reactions vary greatly in different individuals. The toxicity is dependent upon the individual tolerance or sensitivity to the drug.[15,27,36]

Avitaminosis C has caused considerable debate as a possible factor in the production of the lesion. Frankel[12] and Kimball[17] believed a relationship existed between Vitamin C deficiency and the amount of hyperplasia, and that the deficiency of the vitamin parallels the hyperplasia. Drake[8] administered dilantin sodium orally to albino rats. He found an increase in the excretion of ascorbic acid in the urine (maximum after four days), and that the body supply appeared to be gradually decreased by administration of dilantin. Ennever and Pendo[9] found that none out of 40 mice developed hyperplasia when given 60-70 mg. dilantin per kilogram body weight. The injections were made every 24 hours for 7 days. They suggested that the Vitamin C synthesized by the animals prevented the hyperplasia.*

Conversely, other experiments have shown that Vitamin C is not a factor. Putnum,[22] found that avitaminosis C did not exist in the patients developing the hyperplasia. Burket[5] and Millhon and Osterburg[23] have also found this to be true. The Parke-Davis Company found that the Vitamin C content of the blood of rats receiving large doses of dilantin sodium showed no decrease in this vitamin. Other factors ruling out avitaminosis C is the absence of purpura, sore joints, extreme weakness, marginal gingival necrosis, petechial hemorrhages, and periodontal membrane involvements.[25,27,29]

Brandon[4] thinks the cause is probably from secretions of by-products of dilantin sodium from the salivary glands coming in contact with epithelium and thereby causing a proliferation. Clinical evidence points to endocrine disturbances in the background of a great number of cases, but these cannot be clearly identified.[10,27] Salama and Hilmy[28] reported an endocrine disturbance from dilantin sodium. The patient involved, a child, had a muscular torso, precocious sex development, marked pubic hair, and the teeth were covered with a hyperplastic gingiva. No diminution of saliva has been found, therefore dryness does not cause it.[32]

*The diet was not mentioned in this experiment. If self-cleansing this could have been an important factor. See reference number 16.

TREATMENT

One method of treatment is withdrawal of the drug.[1,25,32] Unless the lesions are severe, dilantin should not be stopped.[5] Discontinuance of the drug does not necessarily mean a subsiding of the hyperplasia. Phenobarbital[24] or bromides[26] may be substituted for the dilantin sodium. The proper dosage is the most important factor, and not the withdrawal of the drug, except in exaggerated cases.

Another method of treatment is the gingivectomy. The surgical removal of the tissue should be done if toothbrushing is painful, mastication is impaired, or for cosmetic reasons.[1,10,32] This method is rapid and effective, but unless the patient follows with correct home care treatment rapid recurrence of the hyperplastic tissue will result.[1,32,36,37] Stammers[31] advocates a combination of surgery and x-ray therapy to prevent recurrence. In some of his cases no recurrence took place. It is well to keep in mind that the patient is an epileptic and that nervousness produced by the operation may bring on an attack.[1] In almost all cases, the hyperplasia will recur after its surgical removal.[1,4,32,36]

Another and most satisfactory method of treatment is conservative periodontal treatment.[27,33] This type of treatment is well tolerated by the patient.[1] Even this method does not assure that the hyperplasia will be checked.[4] Sealing, curettage, restoration of carious teeth, and home care methods will eliminate the inflammatory complications.[1,3,4,5,27,33]

Hyperplasia will disappear with full mouth extractions, even with continuance

of dilantin.[16]

The four methods given may be used in combination with one another. "There is little to be gained from local treatment as long as the patient remains upon general treatment with dilantin sodium. If the patient's tolerance for the drug can be established, its use may control the convulsions without causing gingival tissue disturbances.[1,7]

PREVENTION

There should be close cooperation between the dentist and physician before the drug is given to permit the dentist to remove sources of local irritation. This includes scaling and subgingival curettage, general operative procedures to restore contact and contour, replacement of missing teeth, and adjustment of occlusion. The patient should also be instructed in toothbrushing and in improvement of diet, if necessary.[1]

SUMMARY

1. Dilantin sodium is a useful drug in controlling convulsive seizures.

2. In approximately 50% of the cases treated with dilantin sodium, gingival hyperplasia occurs.

3. The exact cause of the hyperplasia is not known.

4. Methods of treatment are:
   a. Withdrawal of the drug
   b. Gingivectomy
   c. Conservative periodontal treatment
   d. Extractions

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Abstracts of Interest to the Periodontist

Gladstone, Sidney A.: **Sponge Biopsy in Office Practice. A New Method in the Diagnosis of Cancer.** *J.A.M.A.* **145 (16):** 1238-1244, (April 21) 1951.

Dr. Gladstone reports the use of this new method of sponge biopsy for cancer diagnosis on over 1200 patients during the past two years. The method is of great value in general practice in diagnosing or detecting cancers of the oral cavity and skin and of the cervix uteri and rectum. It is especially recommended for its availability and its comparative simplicity. A small piece of sponge, cellulose or gelatin, is rubbed firmly across suspected area. "Tissue juices and detached tissue particles" are absorbed by the sponge which is then dropped into a formaldehyde fixing solution and turned over to the pathologist at the operator's convenience. In the laboratory the specimen is processed after the usual method for tissue biopsy specimens. If case is positive, cancer cells and cancer tissue will be noted under microscope. Sponges of both cellulose and gelatin have been satisfactorily used, each possessing slight advantages in certain respects.

Sponge biopsy was used in 40 suspicious oral lesions and 12 were diagnosed as positive. In all cases diagnosis was confirmed by surgical biopsy. Sponge biopsy is very effective and applicable in the more malignant types of oral cancer where a soft friable tissue surface presents with early ulceration. The method is so comparatively simple that it should be used routinely on every small ulcer or nodule which cannot

be explained on the basis of trauma or infection. The following figures indicate the importance of earliest possible diagnosis; in lesions detected when less than 2 centimeters in diameter the five year survival period following treatment is doubled. Some of the chronic mouth lesions do not lend themselves well to sponge biopsy, as when they present a hard surface which does not rub off on the sponge. Such chronic but usually less malignant conditions require deeper surgical specimens for biopsy. The same type of hard non-ulcerating surface is often encountered in skin cancers; here the overlying epidermis may sometimes be curetted and an exudate inducted which may be used with the sponge method. The article discusses at considerable length the use of sponge biopsy for cancers of the rectum and the cervix of the uterus.

In a discussion which followed Gladstone's presentation, Dr. Howard L. Richardson emphasized the advantages of the method from the standpoint of the pathologist. Dr. Emil Novak questioned its desirability in certain uterine cervix cases while admitting that no one method was applicable to all. Dr. Max Cowett felt the method to be valuable to the proctologist. Sponge biopsy is devoid of the danger which removal biopsy entails of stimulating the activity and dissemination of malignancy cells. The principal advantage to the dentist seems to be simplicity of the method which makes possible its use in office practice on any vaguely suspicious case where diagnosis might otherwise be postponed awaiting developments, a delay which might have disastrous effects.

Perleche. *J.A.M.A.* **145 (4)**:274. (January 27) 1951.

Replying to query regarding woman of 39 who has suffered for many years with "cracking at the corners of the mouth," editor says: This condition (perleche) occurs as a result of streptococci or monilia infection, vitamin deficiency, maceration due to overhanging lips or poorly fitted dentures or hypersensitivity to drugs. Condition may be treated with antibiotic agents, antiseptics or fungicidal preparations, and multiple vitamins. Irritating agents should be avoided.

Excessive Salivation. *J.A.M.A.* **145 (15)**: 1222. (April 14) 1951.

In answer to a query concerning relief from excessive salivation and drooling in a patient who has had partial facial paralysis from a cerebral accident, the following are recommended as most effective in reducing secretion of saliva: belladonna, atropine, scopolamine or stramonium. Banthine (50 mg. three times daily) might also be tried.

The Behcet Syndrome. *J.A.M.A.* **145 (9)**: 6 and 7. (March 3) 1951.

Information is requested concerning the etiology and treatment of Behcet's syndrome, which is characterized by indolent ulcers of the vaginal and oral mucosa. Answer: The Behcet syndrome consists of stomatitis, uveitis and ulcers of the genital organs. The etiology is unknown, but a virus is probably responsible for the syndrome. There is no specific treatment for this condition, therefore the symptoms should be treated locally to give relief. Antibiotics should also be tried.

Oral Reactions to Penicillin Tooth Powder. *J.A.M.A.* **145 (15)**:1004. (March 31) 1951.

In a communication to the Journal, Clifford H. Kalb, M.D. decries the current fashionable use of highly potent allergic sensitizers in products available to the public. He cites a case of urticaria medicamentosa due specifically to brushing the teeth with a professional sample of Dentocillin. This tooth powder according to its label contains 500 units per gram of crystalline potassium-penicillin G and the reaction follows the characteristic course seen following penicillin used in the customary manner, urticaria appearing between 48 and 60 hours after initial brushing. C. R.

BETH ISRAEL HOSPITAL

The Periodontia Department of the Beth Israel Hospital of Boston, Massachusetts, announces a course in Periodontia, under the direction of Dr. Henry M. Goldman, from October 6 to 17, 1952. The following is an outline of the material to be covered: Anatomy, Physiology, Functional Changes, Pathology, Periodontal Pocket, Occlusion, Odontexesis—Instrumentation, Subgingival curettage, Gingivectomy, Intrabony pocket, Gingivoplasty, Occlusal equilibration and adjustment, Temporary splinting, Tooth movement (individual), Mouth rehabilitation (splinting), Examination—diagnostic signs and symptoms, Etiology—interpretation to pathology, Diagnosis—case diagnosis: clinical, radiographic, Vincent's infection, Mouth diseases, Case study and handling, Oral hygiene.

Instruction will be carried on by clinics, lectures, demonstrations and motion pictures. Each participant can carry out on patients the clinical procedures, under staff supervision.

For enrollment please contact Director of Dental Department, Beth Israel Hospital, Boston, Mass.

TEMPLE UNIVERSITY ANNOUNCES A COURSE IN CLINICAL OCCLUSION

A course designed to present the fundamentals of occlusion in relation to the physiology and pathology of the periodontal supporting tissues. The entire course will be directed toward correlating the principles of occlusion to clinical criteria and therapeutic endeavors. Includes lectures, seminar conferences and clinical demonstrations by Dr. Lewis Fox, South Norwalk, Conn.

This course will be given June 4, 5, 6, 1952, and is limited to twenty persons. Tuition $100.00.

Applications can be made to:

Dr. Louis Herman
Director of Postgraduate Studies
Temple University School of Dentistry
3223 North Broad Street
Philadelphia 40, Pennsylvania

TWO COURSES IN PERIODONTICS ARE ANNOUNCED BY B. ORBAN FOR JUNE, 1952

1. Theory and Practice of Periodontics—June 9-14

The Theory and Practice course will consist of lectures and demonstrations as follows:

Lectures: The epithelial attachment; Gingival sulcus and pocket
The normal gingiva; Periodontal membrane
Alveolar bone; Cementum
Biology and Pathology of bone
Systemic aspects of Periodontal diseases
Tissue changes due to trauma
The problem of occlusion in periodontal practice

Demonstrations: Prophylaxis; Home-care technique
Gingivectomy, post-operative treatment
Registration of hinge axis; Mounting of casts in articulator
Occlusal adjustment

Class starts Monday, June 9 at 8:30 A.M. and ends Saturday, June 14, at noon.

Fee: $200.00

2. The Problem of Occlusion in a Periodontal Practice—June 20-25

This course will consist of the following:

Lecture: The biology of the supporting tissues of the teeth, with special emphasis on their functional structure and adaptation.

Demonstrations: Determination of the hinge axis of the mandible
Registration of centric and eccentric jaw movements
Proper mounting of casts on an articulator
Adjustment of occlusal disharmony on casts
Adjustment of occlusal disharmony in the mouth of patient

Workshop: Casts will be supplied to each member of the class in order to do occlusal adjustment by trimming them. *Please bring your articulator with you.*

Class starts Friday, June 20 at 8:30 A.M. and ends Wednesday, June 25.

Fee: $200.00

Classes will be held at 629 N. Nevada Ave., Colorado Springs, Colo.

SEVENTH ANNUAL MEETING OF THE NORTHWEST CONFERENCE IN DENTAL MEDICINE

The Northwest Conference in Dental Medicine announces that it will hold its Seventh Annual Meeting April 13th to 17th, 1952, at the Columbia Gorge Resort Hotel, Hood River, Oregon. The Portland Chapter will be host.

This conference is an annual scientific meeting designed to acquaint the dental, medical and allied professions with the newer concepts concerning the etiology, pathology, prognosis and treatment of diseases of the oral cavity. The effect of systemic disorders on oral structures is stressed.

The conference will consist of four days of lectures, seminars and round table discussions of clinical problems by outstanding teachers and clinicians in dentistry, medicine, nutrition, public health, and related fields.

The program has been arranged so that morning and evenings are devoted to professional activities while the afternoons are open for recreation. A program offering special activities for the ladies is scheduled.

Some of the important topics to be analyzed and discussed at this meeting are:

1. Oral infections associated with systemic disturbances.
2. Antibiotic therapy.
   (a) Why — when — where — how.
3. Nutrition from the ground up.
   (a) Agronomy, soil conservation, soil chemistry, danger of insecticides.
   (b) Metabolism of calcium, the formation of tooth and bone structures, factors that interfere with normal calcifying processes.

4. A study of the effects of fluorine on laboratory animals.

5. Oral and systemic effects of fluorine,—absorption, fate excretion and action. A round table discussion pro and con by representatives from the U. S. Department of Public Health, Medical Profession, Dental Profession, and allied groups.

For programs and reservations, address Dr. Cecil V. Ross, Secretary and Treasurer, 829 Medical Arts Building, Portland 5, Oregon.

COLUMBIA UNIVERSITY

Four postgraduate courses in Periodontology are offered at Columbia University, School of Dental and Oral Surgery of the Faculty of Medicine.

A full time program covering a period of two academic years leads to a Certificate of Training in Periodontology. Correlation of the fundamental sciences to clinical practice is emphasized.

Of the three part-time courses, one course will be given one full day a week from September 24, 1952 to May 5, 1953; a second course will be given one half day each week from September 24, 1952 to May 5, 1953; and the third course will be full time from June 2 to June 13, 1952.

Application for admission to these courses should be made not later than May 15, 1952. For further information write to Dr. Barnet M. Levy, Director, Postgraduate Division, School of Dental and Oral Surgery, Columbia University, 630 West 168th Street, New York 32, N.Y.

POSTGRADUATE COURSE IN PERIODONTICS THE OHIO STATE UNIVERSITY COLLEGE OF DENTISTRY

A postgraduate course in Periodontics will be held May 12 to 16, 1952, at the College of Dentistry, Ohio State University, under the direction of Dr. John R. Wilson. The fee for this course is $50.00, and applications may be obtained from the Postgraduate Division of the College of Dentistry, Ohio State University, Columbus 10, Ohio.

THE THIRD MID-ATLANTIC STATES SEMINAR IN ORAL MEDICINE

Announcement is made of the Third Mid-Atlantic States Seminar in Oral Medicine to be offered May 11 to 16, 1952, by the School of Dentistry, University of Pennsylvania. Lecturers will include Dr. Oscar V. Batson, Professor of Anatomy, Graduate School of Medicine, University of Pennsylvania; Dr. William Branstad, Assistant Professor in the Department of Oral Medicine, School of Dentistry, University of Minnesota; Dr. Henry M. Goldman, Head of the Dental Department and Director of Dental Research at the Beth Israel Hospital, Boston, Massachusetts; Dr. Ernest R. Granger, Director of the postgraduate courses in Mouth Rehabilitation, School of Dentistry, University of

Pennsylvania; Dr. Richard S. Manly, Professor of Dentistry and Director of Special Research Projects at Tufts College Dental School; and Dr. Robert E. Moyers, Professor of Orthodontics and Head of this department at the School of Dentistry, University of Toronto.

The fee for the seminar, which includes registration, lodging and meals at Skytop Club where the course will be held, is $250.00. Applications should be mailed to the Director, Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce Street, Philadelphia 4, Pennsylvania.

AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES ANNUAL MEETING

The Sixth Annual Meeting of the American Academy of Dental Medicine will be held at the Mount Royal Hotel, Montreal, Canada, on Thursday, Friday and Saturday, May 29, 30 and 31, 1952. On Wednesday, May 28th, there will be a registered pre-convention seminar of courses on oral medicine, dental pathology, and psychosomatic factors of dental practice.

The regular meeting will further include a series of lectures and round table discussions on dento-medical subjects. A program of sightseeing and entertainment is also being arranged. All members and interested dentists and physicians are cordially invited to attend. Full details as well as reservations for registrants and their families are available through the National Secretary, Dr. William M. Greenhut, 124 East 84th Street, N. Y. 28, N. Y.

The Academy was organized in 1945 "To promote the study and dissemination of knowledge of the cause, prevention and control of disease of the teeth, their supporting structures and appendage parts, and related subjects; and to foster and promote a better scientific understanding between the fields of dentistry and medicine." It has an international membership of about one thousand.

ANNUAL MEETING OF THE AMERICAN ACADEMY OF APPLIED NUTRITION AND THE AMERICAN ANTI-ARTHRITIS ASSOCIATION

The 1952 Annual Meeting of the American Academy of Applied Nutrition and the American Anti-Arthritis Association will be held at the

DEL MAR HOTEL
DEL MAR, CALIFORNIA
April 25, 26, 27

Registration will begin Thursday evening, April 24. The registration fee is five dollars, regardless of membership classification or status as guest, and applies for one meeting or the full three days. The Del Mar Hotel provides rooms and meals for twelve dollars per person per day, including tax and tip.

Joint Report of the Committees on Classification and Nomenclature of the American Academy of Periodontology

Based on the Proceedings of the Workshop on Terminology and Classification of Periodontal Diseases sponsored by the A.A.O.P. and A.A.P. held on Sunday and Monday, October 7 and 8, 1951. The duly appointed representatives of the A.A.P. and the A.A.O.P. met in Washington, D. C. Those who were present are listed below:

A.A.P.—Bernier, Becks, Ray, Leabo, Thomas, Nuckols, McCall, Leonard, Miller, Lyons, Goldman, Beube.

A.A.O.P.—Cahn, Kerr, Wienmann, Hartman, Kesel, Boyle, Glickman, Hill, Hine, Coolidge.

The meeting was called to order at 9:00 a.m., Sunday, October 7, with Colonel Joseph L. Bernier presiding.

The members of the Workshop unanimously recommended that both Academies give serious consideration to the suggestion regarding the establishment of an International Forum to be held in London, in 1952, in conjunction with the F.D.I.

Following the presentation of the formal papers open discussion followed. It was agreed that any classification of periodontal diseases should,

a) Be directed primarily to the clinician.

b) Be based on fundamental processes rather than etiology.

c) That all disturbances of the periodontium be classified under one of the following four fundamental "processes.": 1. Inflammation. 2. Dystrophies. 3. Neoplasia. 4. Anomalies.

It was the unanimous feeling of the members that all disturbances of the periodontium listed under the four major categories (see above) be designated under the term "Periodontal Disease."

The periodontium as referred to in this report was considered to include: a) Gingivae; b) Pericementum; c) Cementum; d) Alveolar bone.

The term pericementum was selected because the word "periodontal," in the phrase, "periodontal membrane, contains the same prefix and noun (periodont) as in the word Periodontium. Such a similarity makes it impossible to suitably designate reactions limited to this structure.

This becomes obvious because the term periodontitis necessarily designates an inflammation of any or all parts of the periodontium, but no one part specifically.

After considerable discussion with the umpires and etymologists it was the sense of those present that the prefixes "para" and "peri" could in the interests of expediency for the present be considered synonymous. The group feel, however, that this report should use the prefix "perio." The written opinions of the etymologists on this point are shown in appendix.

There followed a detailed discussion relating to terminology, however, these discussions did not progress sufficiently to permit the inclusion of positive recommendations in this report.

Summary:

It was enthusiastically agreed that this Workshop accomplished the tremendous task of "cutting through" existing barriers which have impeded the establishment of a fundamentally sound and universal classification of periodontal disease. To this end the basis and medical sciences were reconciled with the problems of periodontology. This correlation resulted in the formation of a suitable framework upon which a scientifically correct classification can be predicated.

The committees on Classification and Nomenclature and the members of the Workshop were outspoken in their belief that this undertaking should be considered only as a preliminary step and should therefore be continued in order that the progress made can be applied to the eventual formulation of a classification and suitable nomenclature.

Recommendations:

It is recommended that the present committees on nomenclature and classification be appointed for another year and that the two Academy's representatives be retained in their present capacity for the same period. In this way the Workshop will be reconvened to continue the progress in this important undertaking. It is also our opinion that if possible the same umpires and etymologists, possibly with others in their field, be secured to attend this proposed Workshop inasmuch as they are familiar with the progress to date.

The Committees on Classification and Nomenclature wish to extend their thanks to General O. P. Snyder and his staff for the excellent facilities provided at the Army Dental School.

It is further recommended that the Council instruct the Secretary to officially thank the umpires and etymologists for their valuable contribution to this Workshop.

JOSEPH L. BERNIER
Chairman, Nomenclature Committee

HERMANN BECKS
Chairman, Classification Committee

Appendix

The group prefers to use the word *periodontium* in its report. This I also prefer because:

1. The Greek prefix *peri,* meaning "around" seems much more specific for the structures *around* the teeth than Greek *para,* which simply means "near" or "beside."

2. By using *peri* one avoids confusion in spelling with Latin *par* meaning "equal" and in pronunciation with Latin *per* meaning "through." With *parodontium* there would be confusion, especially because of the elision between the final *a* of *para* and the *o* of odontium.

3. This elision would be avoided by using the Latin noun with the prefix *para, paradentium.* I can "take" hybrid words when they are necessary, and most of us employ them every day; "television" is an example. But I hesitate then to add as a third item a Greek suffix:

para	- dent	- osis	(or, itis)
Greek	Latin	Greek	

4. I have worked in Europe many years and believe heartily in international cooperation. I do not believe I am "provincial" when, for the reasons given above, I prefer *peri* to *para.*

(sig.) Dorothy M. Schullian

8 October 1951